# The American Journal of Obstetrics and Gynecology

VOL. XII ST. LOUIS, NOVEMBER, 1926 NO. 5


## Original Communications

### STUDIES IN ANESTHESIA, ANOXEMIA, ANHYDREMIA AND ECLAMPSIA, WITH CERTAIN DEDUCTIONS CONCERNING THE TREATMENT OF ECLAMPSIA


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FROM a consideration of the blood changes in eclampsia, as recently reported by Stander and Radelet,[1] it became apparent that one may produce similar changes in animals by the use of anesthesia. The outstanding changes in the blood of an eclamptic woman are: a hyperglycemia, a high uric acid, an increased lactic acid, a low $CO_2$-combining power, and often an elevated inorganic phosphorus. The changes produced in the blood sugar,[2] the $CO_2$-combining power,[3] and the lactic acid[4] under certain of the general anesthetics, suggested a similarity in the blood changes in these two conditions.

#### METHODS

All determinations were made on dogs that had been starved for 18 hours preceding the experiment. In every instance an initial blood specimen was obtained, usually from the saphenous vein, at about nine o'clock on the morning of the experiment. Subsequent blood samples were taken at different intervals as indicated in the various protocols and tables. An endeavor was made to use only dogs that were evidently quite healthy and normal and that had not been subjected to previous experimental work.

The blood constituents studied were the nonprotein nitrogen, urea nitrogen, sugar, uric acid, lactic acid, inorganic phosphorus, and carbon dioxide combining power. Sodium oxalate having been used as an anticoagulant, a Folin-Wu filtrate was made within half an hour after the blood was drawn. A sugar determination was immediately made on a portion of this filtrate by the method of Benedict[5], while another portion was being treated with $CuSO_4$ and $Ca(OH)_2$ in preparation for the lactic acid determination, which was carried out according to the procedure of Clausen[6], sulphuric acid being used for the formation of acetaldehyde from lactic acid. The remaining portion of the Folin-Wu filtrate was then analyzed for nonprotein nitrogen and uric acid by the methods of Folin[7]. The urea nitrogen was determined by the Van Slyke-Cullen modification of the Marshall method[8] and

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the $CO_2$-combining power according to the technic of Van Slyke[9]. Inorganic phosphorus determinations were made by the colorimetric method according to the modifications of Briggs[10]. All values are expressed in mg. per 100 c.c. of blood, except the $CO_2$-combining power, which is written in volumes per cent.

Normally, there is no, or exceedingly little, uric acid in the circulating blood of the dog. By diluting the standard solution it has been possible to obtain a comparison with the color produced in the blood filtrate of the normal dog. It must be pointed out, therefore, that the uric acid figures reported for dogs may not be absolute but they are of distinct value when employed for a comparison under different conditions. These criticisms do not apply to the uric acid values reported for the human.

## PART I. ANESTHESIA

*Ether.*—Ether was administered by inhalation to six dogs. The blood changes were almost identical in the six animals, and Table I shows the typical variations, studied over a period of two hours. The following protocol demonstrates the effect of about half an hour's deep ether anesthesia.

TABLE I

ETHER

| TIME | PROCEDURE | $CO_2$ | SUGAR | URIC ACID | LACTIC ACID | N. P. N. | PHOSPHORUS |
|---|---|---|---|---|---|---|---|
| 9.43 A.M. | Blood specimen. | | | | | | |
| | Ether started. | 59.7 | 125 | 0.5 | 14.5 | 52.2 | 3.0 |
| 10.48 A.M. | Ether stopped. | | | | | | |
| | Blood specimen. | 51.2 | 187 | 0.6 | 14.5 | 52.2 | 3.3 |
| 11.01 A.M. | Blood specimen. | 46.3 | 167 | 0.7 | 19.3 | 45.6 | 3.3 |
| 11.16 A.M. | Blood specimen. | 50.3 | 167 | 0.6 | 19.3 | 46 | 3.3 |
| 11.46 A.M. | Blood specimen | 49.3 | 167 | 0.6 | 16.9 | 46 | 3.3 |
| 12.46 A.M. | Blood specimen | 58.8 | 167 | 0.5 | 15.7 | 46 | 3.0 |

*Dog No. 6.*—Male, weight 25 kg., 4/15/25; 9:50 A.M. First blood sample taken; 9:55 A.M. Ether started; 10:20 A.M. Deep anesthesia attained; 10:55 A.M. Second blood sample taken, while animal was still under deep anesthesia.

ANALYSES OF THE TWO BLOOD SPECIMENS

| | *Before Ether* | *Under Ether* |
|---|---|---|
| $CO_2$ | 63.1 | 41.2 |
| Sugar | 125 | 222 |
| Uric Acid | 0.9 | 1.2 |
| Lactic Acid | 14.5 | 18.1 |
| Phosphorus | 3.1 | 3.8 |
| N. P. N. | 55.0 | 56.0 |

In the ether experiments an attempt was made to induce varying degrees of anesthesia in the different dogs, in order to see whether any parallelism between the depth of anesthesia and the blood changes could be established, and also to observe to what extent asphyxia may play a part in the production of these blood changes. The results show a relationship between the depth of anesthesia and the blood changes produced, while they also indicate that asphyxia may play an important part in very deep ether anesthesia, but certainly not in light anesthesia.

*Chloroform.*—Chloroform was administered to five dogs. The changes produced in the blood constituents are shown in the following protocol as well as in Table II, where the studies are over a period of two hours after the anesthetic was stopped.

TABLE II

CHLOROFORM

| TIME | PROCEDURE | $CO_2$ | SUGAR | URIC ACID | LACTIC ACID | N. P. N. | B. U. N. | PHOSPHORUS |
|---|---|---|---|---|---|---|---|---|
| 10.20 A.M. | Blood specimen | | | | | | | |
| | $CHCl_3$ started. | 59.5 | 139 | 0.5 | 8.4 | 47.9 | 15.3 | 3.0 |
| 11.00 A.M. | $CHCl_3$ stopped. | | | | | | | |
| | Blood specimen. | 56.7 | 182 | 0.6 | 10.8 | 51.3 | 18.1 | 3.5 |
| 11.15 A.M. | Blood specimen | 51.9 | 220 | 0.8 | 14.5 | 46.0 | 19.1 | 3.0 |
| 11.30 A.M. | Blood specimen | 51.9 | 210 | 0.8 | 13.3 | 45.0 | 15.8 | 3.3 |
| 12.00 N. | Blood specimen | 55.7 | 200 | 0.6 | 13.3 | 45.0 | 10.6 | 3.1 |
| 1.00 P.M. | Blood specimen. | 55.7 | 182 | 0.5 | 12.1 | 45.6 | 12.0 | 3.8 |

*Dog No. 12.*—Female, weight 11 kg., 4/22/26; 10:10 A.M. First blood specimen taken 10:20 A.M. Chloroform started; 10:35 A.M. Deep anesthesia; 11:05 A.M. Chloroform stopped; 11:20 A.M. Second blood specimen taken.

ANALYSES OF THE TWO BLOOD SPECIMENS

| | *Before Chloroform* | *After Chloroform* |
|---|---|---|
| $CO_2$ | 53.3 | 49.4 |
| Sugar | 134 | 213 |
| Uric Acid | 0.9 | 1.1 |
| Lactic Acid | 8.5 | 18.1 |
| Phosphorus | 2.0 | 2.5 |
| N. P. N. | 37.5 | 37.5 |

In two dogs death was produced by chloroform, and the blood changes noted were quite similar to, although far more striking than those given in the above protocol.

*Nitrous Oxide.*—The effect of nitrous oxide was studied in two dogs. The first dog died within forty minutes after the anesthesia was started, while in the second animal, there was a chance to observe the blood changes over a period of about three hours. The following protocol and Table III record the changes noted.

TABLE III

NITROUS OXIDE

| TIME | PROCEDURE | $CO_2$ | SUGAR | URIC ACID | LACTIC ACID | N. P. N. | PHOSPHORUS |
|---|---|---|---|---|---|---|---|
| 9.20 A.M | Blood specimen. | | | | | | |
| | $N_2O$ started. | 56.0 | 121 | 0.5 | 8.5 | 32.8 | 2.5 |
| 9.40 A.M. | $N_2O$ stopped. | | | | | | |
| | Blood specimen | 41.9 | 189 | 0.5 | 15.7 | 33.9 | 2.5 |
| 9.55 A.M. | Blood specimen | 43.8 | 182 | 0.7 | 31.4 | 38.7 | 2.5 |
| 10.10 A.M. | Blood specimen. | 47.5 | 200 | 0.5 | 31.4 | 37.5 | 2.8 |
| 10.50 A.M. | Blood specimen. | 52.2 | 200 | 0.5 | 14.5 | 34.1 | 2.8 |
| 11.50 A.M. | Blood specimen | 52.2 | 189 | 0.5 | 13.3 | 31.6 | 3.2 |

*Dog No. 10.*—Female, weight 10 kg., 4/20/26; 9:30 A.M. First blood specimen taken; 9:45 A.M. Nitrous oxide started. Deep anesthesia almost immediately; 10:20 A.M. Second blood specimen taken, just before death; 10:21 A.M. death.

ANALYSES OF THE TWO BLOOD SPECIMENS

| | *Before* $N_2O$ | *After* $N_2O$ |
|---|---|---|
| $CO_2$ | 51.9 | 41.9 |
| Sugar | 143 | 299 |
| Uric Acid | 0.6 | 1.0 |
| Lactic Acid | 9.6 | 27.7 |
| Phosphorus | 2.3 | 3.0 |
| N. P. N. | 50.0 | 51.4 |

It is my opinion that there was a certain amount of asphyxia just before death, and thus the changes recorded in Table III represent perhaps more accurately the effect of nitrous oxide.

*Ethylene.*—The following protocol is that of a dog to which ethylene gas was administered by two trained anesthetists from this hospital.

*Dog No. 23.*—Male, weight 14 kg., 5/7/26; 10:00 A.M. First blood specimen taken; 10:40 A.M. Ethylene anesthesia started; 11:40 A.M. Second blood specimen taken after twenty minutes of deep anesthesia.

ANALYSES OF THE TWO BLOOD SPECIMENS

| | *Before* $C_2H_4$ | *After* $C_2H_4$ |
|---|---|---|
| $CO_2$ | 54.2 | 51.6 |
| Sugar | 98 | 125 |
| Uric Acid | 0.6 | 0.8 |
| Lactic Acid | 17.5 | 27.7 |
| Phosphorus | 1.7 | 2.5 |
| N. P. N. | 52.4 | 39.2 |
| Urea Nitrogen | 19.6 | 19.6 |

From the above protocols and tables, it will be seen that all four general anesthetics used produced about the same changes in the blood chemistry of the dog. These changes may be stated as follows:

(a) A lowering of the $CO_2$-combining power.
(b) A hyperglycemia.
(c) An increase in lactic acid.
(d) A slight increase in uric acid.
(e) A slight increase in inorganic phosphorus.
(f) No or very little disturbance in the nonprotein and urea nitrogen.

*Anoxemia.*—In the experiments with general anesthesia, it became apparent that often one had to deal with varying degrees of asphyxia. For comparison, I, therefore, subjected a dog to breathing an atmosphere containing 7 per cent oxygen. This was done by taking three Douglas bags, filling two with nitrogen and one with room air. The three bags were connected with one another, forming one large reservoir, with an oxygen content of about 7 per cent. A tight fitting mask, as previously described[11] was placed over the muzzle of the dog and connected by means of a Louven valve to the reservoir in such a manner that the dog inspired air containing 7 per cent oxygen. The animal was subjected to this type of breathing for half an hour, and the following blood changes were noted:

ANALYSES OF THE TWO BLOOD SPECIMENS

| | *Before breathing 7% $O_2$* | *After breathing 7% $O_2$* |
|---|---|---|
| $CO_2$ | 51.9 | 40.4 |
| Sugar | 125 | 330 |
| Uric Acid | 0.6 | 0.8 |
| Lactic Acid | 15.0 | 54.9 |
| Phosphorus | 1.5 | 1.6 |
| N. P. N. | 38.7 | 34.1 |
| Urea Nitrogen | 15.8 | 16.4 |

As will be seen from these figures, the changes produced are almost identical with those recorded above for the general anesthesias. At this point it became necessary to know to what extent lack of oxygen or anoxemia may be a factor in producing those profound blood changes seen under general anesthesia. It seemed plausible that one could eliminate the factor of asphyxia, or at least limit it to a minimum by administering the anesthetic *per rectum* and in such amounts as not to interfere with the rate and depth of respiration. To two dogs ether was given *per rectum* according to the technic of Gwathmey[12]. Both animals showed the typical anesthesia blood changes and the following protocol represents a very light Gwathmey ether anesthesia.

*Dog No.* 28.—Female, weight 16 kg., 5/18/26; 9:00 A.M. First blood specimen taken; 9:10 A.M. Mixture of 65 per cent ether and 35 per cent olive oil introduced into rectum. Amount used about 9 c.c. per kilogram. A light anesthesia produced. 10:10 A.M. Second blood specimen taken.

ANALYSES OF BLOOD SPECIMENS

| | *Before Gwathmey* | *After Gwathmey* |
|---|---|---|
| $CO_2$ | 49.4 | 44.8 |
| Sugar | 91 | 189 |
| Uric Acid | 0.9 | 0.9 |
| Lactic Acid | 15.1 | 18.7 |
| Phosphorus | 2.1 | 1.7 |
| N. P. N. | 45.6 | 43.6 |
| Urea Nitrogen | 18.0 | 18.5 |

In neither dog was there an accumulation of uric acid in the blood, although both showed a lowered $CO_2$-combining power, a hyperglycemia and an increased lactic acid under Gwathmey anesthesia. It is interesting to note that in both dogs the inorganic phosphorus changed definitely but in opposite directions; in the first animal the anesthesia produced a marked elevation in the phosphorus, while in the second, a lowering took place. The nonprotein nitrogen and urea nitrogen were not affected.

*Hypnotics.*—As indicated above, general anesthetics,—ether, chloroform, nitrous oxide and ethylene—all produce similar changes in the blood chemistry of the dog; the changes from ether being the most marked and those from ethylene the slightest. A hyperglycemia, a lowering of the carbon-dioxide combining power, an increased lactic acid content and a tendency towards uric acid accumulation, and elevated inorganic phosphorus values are the typical changes observed. It next became necessary to see what, if any, variations in the blood chemistry follow the use of hypnotics, and for this purpose I used morphine, chloral, chloralose, urethane, paraldehyde, veronal, and amytal. In this part of the work an attempt was made to give such doses of these hypnotics as would produce satisfactory anesthesia

without killing the animal, as recommended in Sollman's "Manual of Pharmacology." The procedure of obtaining blood specimens was the same as used for the general anesthetics.

In Table IV are given the results of analyses on blood specimens obtained before and after the administration of each of the eight hypnotics above mentioned. Three experiments were performed with morphine, two with amytal or isoamylethyl barbituric acid, and one with each of the other drugs; the latter evidently need corroboration. The outstanding finding is the hyperglycemia produced by all these hypnotics, except in the case of amytal, where the blood-sugar change is less than 10 mg. per c.c. of blood. With chloral and chloretone the

TABLE IV

HYPNOTICS

| DRUG | SAMPLE | $CO_2$ | SUGAR | UREIC ACID | LACTIC ACID | N. P. N. | B. U. N. | PHOSPHORUS |
|---|---|---|---|---|---|---|---|---|
| Morphia | Before | 57.5 | 108 | 0.8 | 13.2 | 48.0 | 18.7 | 2.5 |
| | After | 70.0 | 167 | 0.8 | 11.5 | 40.5 | 18.7 | 1.7 |
| Chloral | Before | 62.6 | 133 | 0.6 | | 33.3 | | 2.4 |
| | After | 53.2 | 119 | 0.6 | | 30.9 | | 2.4 |
| Chloretone | Before | 54.1 | 94 | 0.6 | | 33.3 | | 2.4 |
| | After | 48.5 | 115 | 0.7 | | 23.4 | | 2.6 |
| Chloralose | Before | 59.3 | 103 | 0.5 | | 53.6 | | |
| | After | 52.6 | 233 | 0.5 | | 50.0 | | |
| Urethane | Before | 50.4 | 106 | 0.5 | | 33 | | 2.4 |
| | After | 51.3 | 182 | 0.6 | | 40 | | 2.3 |
| Paraldehyde | Before | 62.6 | 110 | 0.5 | | 37.5 | | 2.3 |
| | After | 45.8 | 169 | 0.6 | | 35.7 | | 1.9 |
| Veronal | Before | 53.2 | 118 | 0.6 | | 35.3 | | 2.6 |
| | After | 52.2 | 144 | 0.7 | | 35.9 | | 2.5 |
| Amytal | Before | 55.1 | 107 | 0.5 | 11.5 | 45.3 | 19.2 | 2.5 |
| | After | 60.0 | 115 | 0.5 | 18.3 | 42.9 | 20.1 | 2.8 |

elevation in blood sugar is also very slight, while with the other soporifics the increase in blood sugar is marked. There is a tendency for the $CO_2$-combining power to be lowered, although we notice a definite rise with morphine, as well as a slight rise with urethane and amytal. To what extent loss of hydrochloric acid through vomiting may play a part in the production of this change in the direction of an alkalosis, is not clear. However, in one experiment with morphine, there was no vomiting, yet the $CO_2$ slightly increased. The other blood constituents showed only insignificant changes.

## PART II. ANHYDREMIA

The work of Volhard and his associates[13] on hypertension and higher protein-split products as well as that of Hashimoto,[14] Hofbauer[15] and others on histamine suggested the study of the blood constituents under certain of these intoxicants. Witte's peptone, histamine, and histamine-free albumose were used in these experiments.

The variations observed are recorded in Table V, and are strikingly similar with the three substances used. There is an increase to almost the same extent in sugar, uric acid, lactic acid, inorganic phosphorus and nonprotein nitrogen, and a nearly identical percentage decrease in $CO_2$-combining power in all three experiments.

It seems fairly evident that the picture resulting from any one of these three substances is partially that of blood concentration; in other words, that the moisture content of the blood is lowered. Hashimoto[15] obtained an increase of solids in the blood stream of from 6 to 17 per cent in acute histamine intoxication. In a healthy dog weighing 12 kg., I injected intravenously 50 mg. of histamine and found that the moisture of the whole blood fell from 82.5 per cent to 80.0 per cent within fifteen minutes. The blood moisture was determined by the technic described by Stander and Tyler.[16] Dale and Laidlaw[17] showed that the increased concentration of the blood

TABLE V

| BLOOD CONSTITUENTS | DOG NO. 18 PEPTONE (0.5 GM. PER KG. INTRAVENOUSLY) | | DOG NO. 17 HISTAMINE (30 MG. INTRAVENOUSLY IN 1:1000 SOLUTION) | | DOG NO. 19 ALBUMOSE "A" (1.0 GM. PER KG. INTRAVENOUSLY) | |
|---|---|---|---|---|---|---|
| | Before | After | Before | After | Before | After |
| $CO_2$ | 49.4 | 29.6 | 54.5 | 34.1 | 50.2 | 27.1 |
| Sugar | 98 | 174 | 89 | 182 | 103 | 137 |
| Uric Acid | 0.8 | 1.1 | 0.7 | 1.0 | 0.6 | 1.0 |
| Lactic Acid | 18.11 | 30.18 | 19.3 | 36.2 | 24.2 | 91.7 |
| Phosphorus | 1.4 | 2.5 | 2.8 | 3.1 | 1.5 | 1.7 |
| N. P. N. | 55.9 | 93.6 | 44.7 | 54.6 | 36.1 | 64.6 |

in histamine shock is not due simply to dehydration of the blood, but to actual loss of plasma resulting from capillary dilatation. The work of Hashimoto suggests that there are various factors causing the increase in nonprotein nitrogen in the blood in acute histamine poisoning. A lowered excretion of nitrogenous substances in the urine as well as increased protein destruction may be among the causative factors. The significance of the blood changes produced with peptone, histamine, and histamine-free albumose will be discussed after the pathologic lesions following their use have been described.

## PART III. PATHOLOGY

Most of the animals were sacrificed shortly after the experiment in order to study the tissues microscopically. We found that the quickest and easiest method of killing the dog was a stab through the heart, the animal dying within half a minute. All tissues were immediately placed in 10 per cent formalin and put through the usual histologic procedures. In some instances, it became necessary to make fat-stain preparations.

The following histologic description of the tissues studied is taken from the notes of Dr. J. W. Williams, who was kind enough to examine the sections for me. In most instances we studied the liver, kidneys, heart muscle, pancreas, spleen, adrenals and lungs, but in the following description we give only the abnormal findings.

*Ether.*—Ether was administered to one dog for half an hour, and the animal was killed three hours later. The liver showed slight fatty degeneration, extending throughout the entire lobule, but there was no necrosis of liver cells. The kidney showed very moderate degenerative changes in the cells of the convoluted tubules.

*Chloroform.*—The effect of chloroform was studied in three dogs, and all showed the typical liver lesions.

*Nitrous Oxide.*—In two dogs, following the administration of nitrous oxide, there were moderate degenerative changes in the center of the lobule. These changes

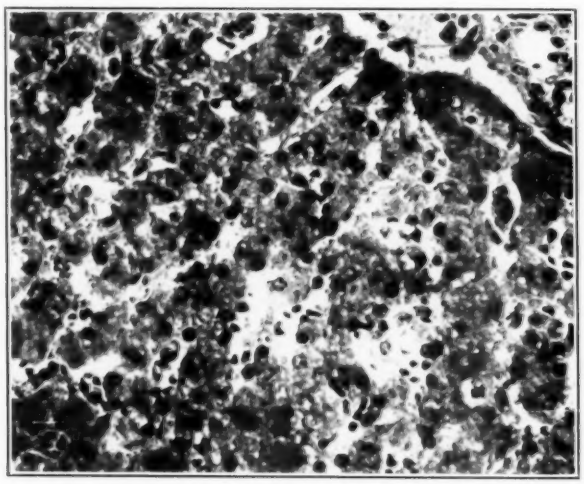

Fig. 1.—Section showing lobular changes in liver after nitrous oxide (x365).

are shown in Fig. 1. Fat stains were positive in the one and negative in the other dog.

*Ethylene.*—The effect of ethylene was observed in one dog. The liver cells showed slight degenerative changes throughout the lobule except at its periphery. Sudan III stain was positive. The kidney sections were normal.

*Gwathmey Anesthesia.*—To one dog ether was administered per rectum, as described above. The liver and kidneys revealed nothing abnormal. A Sudan stain for fat was negative for both the liver and the kidney.

*Histamine.*—We studied two dogs with acute and two with chronic histamine poisoning. All four animals showed marked degenerative changes in the liver and kidney. Except for a few cells at the periphery of the liver lobule, all of the cells showed marked changes. The protoplasm presented a honeycomb structure with a thin unchanged margin. The nuclei in general were well preserved, though in a certain number of cells they were lacking. The degenerative changes in the kidney were restricted to the convoluted tubules. Fat stains showed discrete rounded deposits of fat in all parts of the liver lobule, as well as in the convoluted tubules of the kidney. Fig. 2 demonstrates the changes in the liver.

*Peptone.*—One dog with acute and one with chronic peptone poisoning were studied. Both showed marked changes in the central portion of the liver lobule, not unlike those observed with histamine. There were also degenerative changes in the kidney tubules. Fig. 3 demonstrates the changes in the liver.

*Albumose.*—Two dogs received albumose injections, which produced marked changes throughout the liver lobule quite similar to those following histamine or

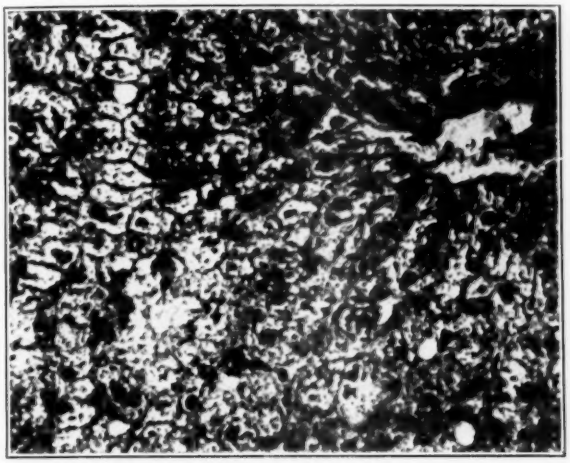

Fig. 2.—Liver changes after chronic histamine poisoning (x400).

Fig. 3.—Liver changes after chronic peptone poisoning (x330).

peptone. The kidney changes were also very marked and of a destructive character restricted to the convoluted tubules.

*Magnesium sulphate.*—Two dogs, following magnesium sulphate injections, had marked fatty changes in the liver, as shown in Fig. 4. This section also shows numerous dilated capillaries and many spaces filled with blood. There were no, or only slight changes in the kidneys.

*Morphine.*—The effect of morphine was studied in one dog. Practically no changes were observed either in the liver or the kidney.

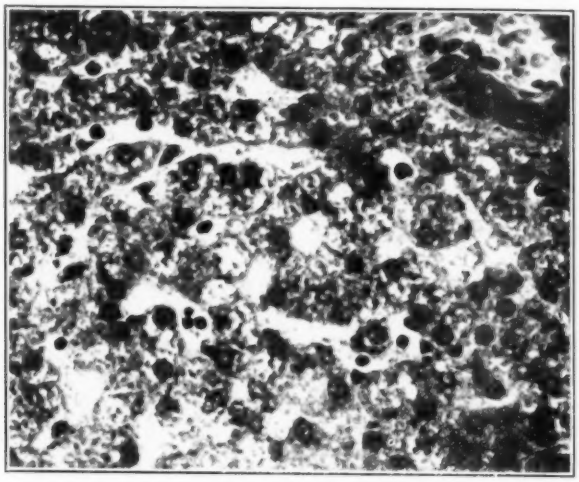

Fig. 4.—Fatty changes in liver after magnesium sulphate injections (x175).

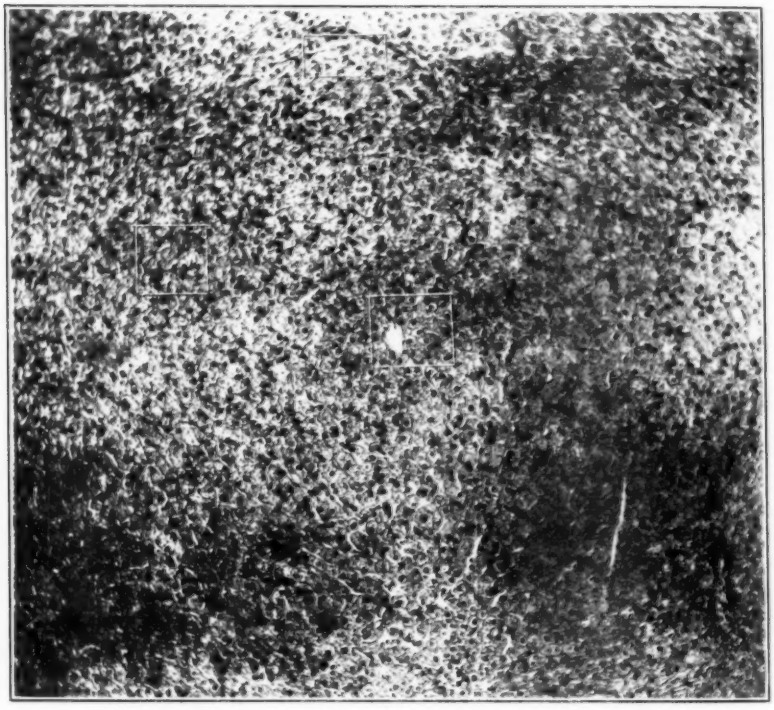

Fig. 5—Changes in liver from anoxemia. (x150).

*Chloretone.*—One dog under chloretone showed no changes in the liver or the kidney.

*Chloral.*—One dog showed marked congestion of the liver, although a fat stain was negative.

*Veronal, Urethane, and Paraldehyde.*—Each of these substances produced marked generalized changes in the liver, whereas the kidney sections were quite normal.

*Anoxemia.*—The changes produced in the liver during the anoxemia experiment described above, are shown in Figs. 5, 6, 7 and 8. There were only slight changes in the kidney. Practically the entire liver lobule was involved. In the center, the cells presented a honeycomb appearance with a narrow peripheral margin of normal

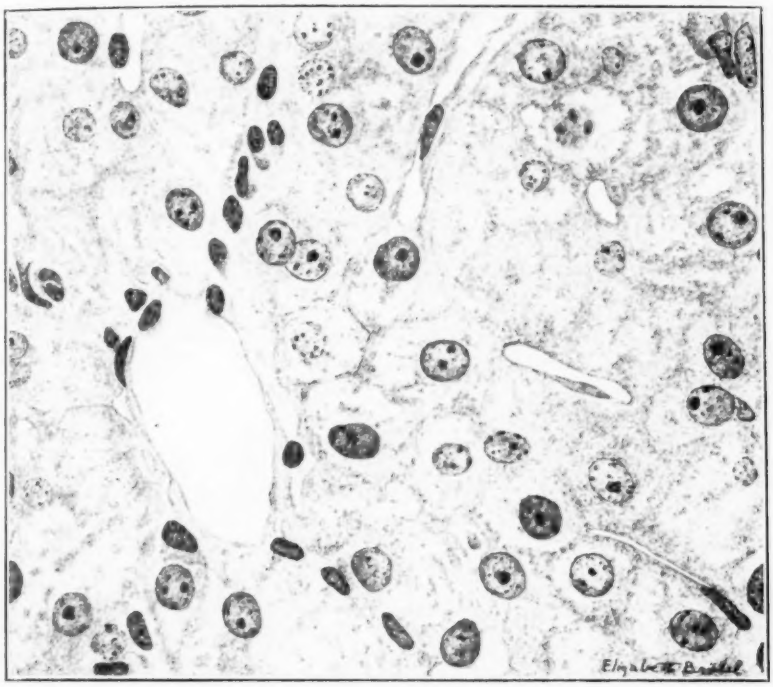

Fig. 6.—Anoxemia. Area around central vein as shown in Fig. 5. (x800.)

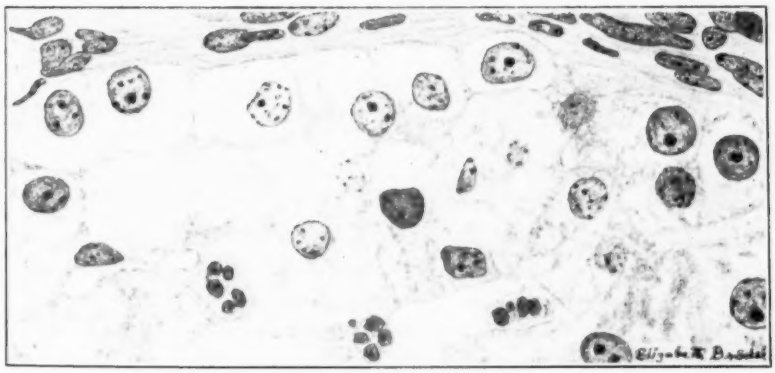

Fig. 7.—Anoxemia. Area near portal space as shown in Fig. 5 (x800.)

protoplasm. Many cells had lost their nuclei and appeared to be destroyed. At the periphery of the lobule a narrow margin of cells was better preserved, but still presented the same type of damage.

While it has been known for years that the prolonged administration of chloroform is occasionally followed by death, when autopsy

shows that it is associated with profound lesions of the liver, it has not been realized that similar, but less extensive, changes occur after the administration of the other general anesthetics, namely, ether, nitrous oxide, and ethylene. Our investigations, which we admit are incomplete, show that marked fatty changes occur in the central portion of the liver lobule, or throughout its entire extent in dogs which have been anesthetized by these agents for so short a period as fifteen minutes, and that the changes are least pronounced when ethylene is employed. Moreover, it should be noted that degenerative changes

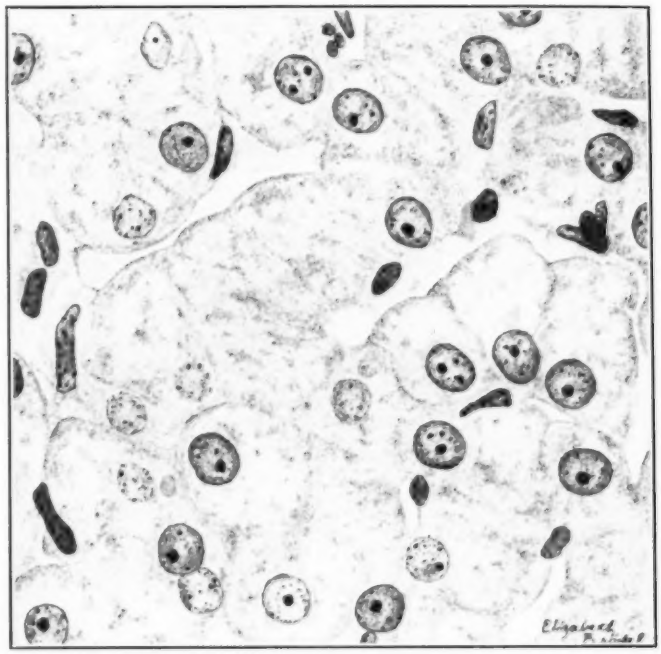

Fig. 8.—Anoxemia. Area near periphery of lobule as shown in Fig. 5. (x900.)

likewise occur in the epithelium of the convoluted tubules of the kidney, and were absent only when ethylene was used.

We cannot assert that similar changes occur in human beings, but reasoning by analogy it is likely, and for that reason the histologic lesions reinforce the deduction drawn from the chemical study of the blood that greater caution should be exercised in the administration of general anesthesia, and particularly in eclampsia.

It should be strongly emphasized that the lesions observed are in no way analogous to those characterizing eclampsia, nor do we make any claim that they are directly responsible for the blood changes described in this study. They are, however, the result of the anesthetic, and it would in general appear inadvisable to employ any

agent in the treatment of eclampsia which we know will lead to still further injury of the already damaged liver and kidneys.

It is of interest to note that in the single instance in which ether was administered *per rectum* by Gwathmey's method, no lesions were observed, though it must be admitted that further observation is necessary before this can be accepted as the general rule. Moreover, it is important to note that the use of magnesium sulphate was associated with the deposition of fat in the liver in large droplets, as well as by slight injury to the kidneys. The former changes, however, were distinctly localized, but were in no way similar to the generalized changes following the use of general anesthesia, or of the various substances to be mentioned later.

When histamine was administered in such large doses as to lead to death, the degenerative changes in the liver and kidney were slight; whereas, when it was administered in small doses for eight or ten days, marked degenerative lesions were produced. These involved the greater part of the lobule, and left free only a rim of cells about its periphery; they closely resembled the changes sometimes observed in fatal cases of vomiting of pregnancy. That the change was in great part fatty was abundantly proved by preparations stained with Sudan III. Marked changes likewise occurred in the epithelium of the convoluted tubules of the kidney. Too much stress, however, cannot be laid upon the fact that the hepatic lesions bear no likeness to those observed in eclampsia.

As one might suppose, *a priori*, identical changes followed the use of peptone and albumose.

It was a matter of surprise that in the anoxemia experiment the entire liver showed signs of marked change in the way of degeneration and necrosis of cells in all parts of the liver lobule, as well as minor changes in the convoluted tubules. As the tissue failed to stain with Sudan, it seems fair to assume that the change was not associated with the deposition of fat, and for that reason it must differ from those thus far described. Resnik and Keefer, in their anoxemia experiments, also produced anatomic changes in the liver, similar to those seen in chronic passive congestion.[18] The lesion following the use of the various hypnotics is of great importance, but as hypnotics were administered to only a single animal in each instance, it would be hazardous to attempt to draw binding conclusions as to their histologic action.

Morphia and chloretone alone gave rise to no lesions; chloral called forth intense congestion, with no serious cellular injury; while veronal, urethrane and paraldehyde all gave rise to pronounced lesions in the liver and to very slight ones, or none at all, in the kidney. In the former, the changes were generalized, and frequently

led to the death of the cells, but they did not appear to be associated with the deposition of fat as was shown by the negative results with specific fat stains.

PART IV. ECLAMPSIA

The changes observed in the blood of eclamptic patients are shown in Table VI, which gives the averages for analyses made during the past year in this Clinic. A comparison between the findings for normal pregnancy at term and for eclampsia reveals the hyperglycemia, the acidosis, and the increase in uric and lactic acids in the latter condition. In order to bring out the marked similarity between

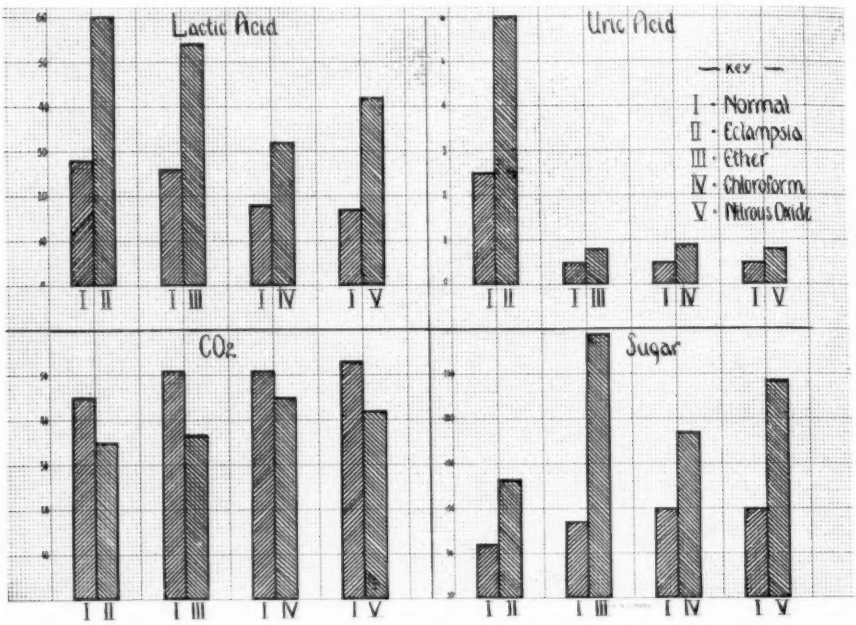


Fig. 9.

the blood changes in eclampsia and in general anesthesia, Fig. 9 has been constructed. The average values for six experiments with ether, five with chloroform, and two with nitrous oxide, as well as the figures in Table VI, form the basis for this graph.

TABLE VI

ECLAMPSIA

| CONSTITUENT | NORMAL PREGNANCY | ECLAMPSIA |
|---|---|---|
| N. P. N. | 28.7 | 34.8 |
| B. U. N. | 13.2 | 13.4 |
| Uric Acid | 2.8 | 6.3 |
| Sugar | 98 | 155 |
| Lactic Acid | 30 | 91 |
| Phosphorus | 2.7 | 3.5 |
| $CO_2$ | 44.7 | 35.2 |

The obvious deduction from this similarity in the blood changes in eclampsia and in general anesthesia is that the condition of the eclamptic patient may be seriously aggravated by superimposing upon an already existing toxemia another due to general anesthesia. Furthermore, it seems quite logical to assume that the better results following the conservative treatment of eclampsia as compared with radical treatment, such as accouchement forcé and cesarean section, are due not to the change in treatment but rather to the fact that fewer eclamptic women are subjected to anesthesia in the conservative technic.

Thalhimer[19] introduced the use of insulin for combating postoperative acidosis as well as vomiting of pregnancy. This therapy has also been extended to include the acidosis of eclampsia[20] in this hospital. I have conducted eight experiments with general anesthesia followed by insulin. The changes observed in all these experiments were similar, whether insulin followed the administration of ether, chloroform,

TABLE VII

ETHER-INSULIN

| CONDITION | $CO_2$ | SUGAR | URIC ACID | LACTIC ACID | PHOSPHORUS | N. P. N. |
|---|---|---|---|---|---|---|
| Normal | 63.1 | 125 | 0.5 | 14.5 | 3.1 | 55.0 |
| After ½ hour Ether | 41.2 | 222 | 0.8 | 18.1 | 3.8 | 56.0 |
| 15 minutes after Insulin | 45.3 | 210 | 0.7 | 13.3 | 3.0 | 54.0 |
| ½ hour after Insulin | 47.1 | 182 | 0.5 | 8.7 | 3.0 | 54.6 |
| 1 hour after Insulin | 50.5 | 113 | 0.5 | 6.1 | 3.1 | 54.1 |
| 2 hours after Insulin | 52.0 | 48 | 0.5 | 6.0 | 3.0 | 54.7 |
| Remarks | Ether was stopped after one-half hour and the dog received immediately 100 units of insulin, intravenously. | | | | | |

or nitrous oxide. A typical ether-insulin experiment is reported in Table VII, in which one sees that insulin not only relieves the acidosis and hyperglycemia, but has also a marked effect on the lactic acid content of the blood. This effect of insulin is similar to that reported by Stander and Radelet[1] in the treatment of eclampsia. It must be emphasized, however, that where insulin is employed to treat the acidosis of eclampsia, one should have definite information regarding the blood sugar and carbon dioxide combining power, and should always give a protective dose of glucose amounting to approximately 2 grams per unit of insulin.

During the past year, magnesium sulphate has been advocated for the treatment of eclampsia.[21] For this reason it seemed necessary to study the effect of this substance on the blood constituents. Two dogs were accordingly given injections of $MgSO_4$, and the results are reported in Table VIII. The only changes observed are a lowering of the $CO_2$-combining power and a slight increase in the blood sugar. From the evidence so far adduced, and the histologic findings already referred to, the use of $MgSO_4$ in eclampsia seems dubious, and it ap-

TABLE VIII

MAGNESIUM SULPHATE

| BLOOD CONSTITUENTS | DOG NO. 32 50 C.C. 50 PER CENT $MgSO_4$ SUBCUTANEOUSLY | | DOG NO. 33 6 C.C. 50 PER CENT $MgSO_4$ INTRAVENOUSLY | |
|---|---|---|---|---|
| | Before | After | Before | After |
| $CO_2$ | 63.6 | 55.1 | 61.5 | 54.1 |
| Sugar | 102 | 147 | 105 | 127 |
| Uric Acid | | | 0.5 | 0.5 |
| Lactic Acid | 30.1 | 41.8 | 12.1 | 14.4 |
| Phosphorus | 2.5 | 2.5 | 2.5 | 2.5 |
| N. P. N. | 40.6 | 40.6 | 50.0 | 51.1 |

pears to me imperative that more experimental work should be done in order to determine the value of this substance in the treatment of eclampsia.

Stroganoff[22] advocates the use of morphine in treating eclampsia. In this connection, it is interesting to observe the blood changes produced by morphine, as reported in Table IV. Morphia has a definite tendency to raise the $CO_2$-combining power, and it may be due to this fact that the Stroganoff treatment is of value in the milder types of eclampsia. Hjort and Taylor[23] as well as Gauss[24] also demonstrated an increase in alkali reserve with morphine.

## PART V. DISCUSSION

*Anesthesia and Eclampsia.*—We have noticed an acidosis and a hyperglycemia with the general anesthetics studied above. The effect of anesthesia on the acid-base balance of the blood has received a great deal of attention, and the experimental evidence at the present time strongly suggests that the same factors are not involved with each of the anesthetics. Ether and chloroform produce a fall in $P_H$ and alkali reserve, leading to an uncompensated alkali deficit, which is probably not dependent on respiratory variations and which, according to Leake,[3] is not caused by formation of ketone bodies or lactic acid. Leake states that a ketosis may develop several hours later as a result of deranged carbohydrate metabolism, related perhaps to a depressing action of ether or chloroform on insulin secretion. In this connection it is well to call to mind the work of Ross and Davis, on the part played by the pancreas in the production of ether-hyperglycemia. These investigators call particular attention to the influence of adrenalin, of sympathetic nerve endings, and of pancreatic secretion on the stored glycogen. While adrenalin and the sympathetic nerve endings influence the liberation of dextrose from glycogen, the pancreatic secretion has an inhibitory effect, i.e., prevents the liberation of dextrose from glycogen. The experimental work of these authors leads them to the conclusion that ether reduces the activity of the pancreatic secretion, resulting in a hyperglycemia, and

that chloroform, while having this same effect, in addition does injury to the liver cells, which would result in not so great a hyperglycemia.

On the other hand, there is evidence[25] suggesting that increase in blood or tissue acidity may produce a greater susceptibility toward a hyperglycemia. Koehler[26] has shown that the ingestion or intravenous injection of acids or acid-producing substances, resulting in a simple acidosis, has a marked effect on the fasting blood sugar of normal human beings. A hyperglycemia of 30 to 40 mg. per 100 c.c. of blood above the normal level follows a drop in $P_H$ from 7.40 to 7.20. The author believes that the depressing effect of the acidosis upon the sugar metabolism may be related to "an acceleration of the inactivation of insulin by glucose due to increased H-ion concentration, especially when there is an overproduction or mobilization of the latter." In how far, then, the hyperglycemia following ether or chloroform anesthesia may be dependent on the initial acidosis or on its effect on the pancreatic secretion is as yet not quite clear. Mann[2] believes that the ether hyperglycemia depends wholly on the liver, as it does not occur when that organ is removed, and furthermore, that it depends chiefly on an intact portal circulation. He showed that ether also depresses the secretion of bile.

The recent work of Ronzoni and her coworkers[4] indicates that the acidosis of ether anesthesia may in a large part be due to the accumulation of lactic acid, rather than itself being the cause of lactic acid production as stated by Anref and Cannan.[27] It is possible, then, that the accumulation of lactic acid, due, say, to asphyxia, may result in an acidosis which in turn may lead to a hyperglycemia.

It seems well established that anesthesia with nitrous oxide cannot be produced unless we have a certain amount of anoxemia. The normal arterial blood is over 92 per cent saturated with oxygen at ordinary oxygen tensions inspired, while, according to Greene,[28] in nitrous oxide anesthesia, the arterial blood is less than 80 per cent saturated with oxygen. Anoxemia evidently then plays an important rôle in the production of the acidosis and hyperglycemia of nitrous oxide anesthesia. It is probable that with ethylene gas we also have to contend with a certain degree of anoxic anoxemia, although more oxygen can be given with this gas than with nitrous oxide.

Koehler, Brunquist, and Loevenhart[29] produced a rapid uncompensated alkali deficit by means of anoxemia. Our figures on anoxemia show that the carbon dioxide combining power dropped over 10 volumes per cent within half an hour of deficient oxygen breathing. This acidosis was accompanied by a marked accumulation of lactic acid in the blood stream together with a profound hyperglycemia.

Various theories have been advanced to explain the action of narcotics. The asphyxial theory of Verworn,[30] the lipoid theory of Meyer and Overton,[31] the cohesive theory of Traube, and the colloid

chemical theories have received a great deal of attention. The lipoid and cohesive theories explain perhaps better the method of transportation than the mode of action of these drugs. It is probable that in general anesthesia we have to deal to a greater or lesser extent with a deoxygenating process. Greene[28] suggests that "the high solubility of nitrous oxide gas in some way interferes with the availability of oxygen, but whether this effect may be attributed to a tissue differential solubility, as in the case of ether, is still under investigation." In this connection, it may be well to note the effect of anesthesia on the lactic acid content of the blood. Our figures show a definite increase of blood lactic acid following the administration of anesthesia, and it is well established that deficient oxidation processes lead to an accumulation of lactic acid in the blood.[32]

It is interesting to observe that even with amytal, where we found only very slight changes in the blood sugar, recent work indicates that this substance has some effect on the metabolism of carbohydrates. Hines, Boyd, and Leese[33] showed that amytal anesthesia lessened the ability of an animal to handle glucose when injected by the continuous intravenous method. An animal so treated showed an increased hyperglycemia and glycosuria, associated with a slight lowering of the $P_H$ of the plasma. They conclude that it is not safe to assume that amytal is associated with no alteration of carbohydrate metabolism.

The blood picture in eclampsia is so similar to that observed under anesthesia, that one wonders whether insufficient oxidation does not play a rôle in eclampsia. The idea seems quite plausible that any theory which tends to explain the causation of eclampsia should be linked up in some way or other with deficient oxidation.

*Anhydremia and Eclampsia.*—It should be stated at the outset that in eclampsia the blood is not more concentrated than in normal full-term pregnancy; on the contrary the work of Stander and Tyler[16] on blood moisture showed that in eclampsia the percentage of water in the blood stream may be great enough to constitute a true hydremia.

The above-recorded results obtained with peptone, albumose, and histamine indicate that these substances produce an anhydremia which in great part explains the blood changes observed. It must also be noted that these substances produce similar liver lesions, wholly different, however, from the eclamptic liver lesion. The liver lesions observed under peptone, albumose, or histamine poisoning are very similar to those seen in fatal vomiting of pregnancy, but before one can assume a connection between any of these substances and vomiting of pregnancy, it will be necessary to determine exactly what rôle starvation may play in the liver lesion of the latter condition, as well as to observe the effect of these substances when given in small amounts to animals throughout the course of pregnancy. It will, fur-

thermore, be necessary to study the blood in vomiting of pregnancy with particular reference to these substances.

The blood pressure in eclampsia[34] is decidedly elevated above the normal, whereas with peptone, albumose, and histamine,[35] we observe a fall in blood pressure. Nevertheless, although eclampsia is associated with a high blood pressure, a tendency towards a hydremia and peripheral necrosis in the liver, while the three substances under discussion produce a fall in blood pressure, an anhydremia and a characteristic liver-lesion which is not peripheral, we feel that from the work of Volhard and his associates[13] it is imperative that further investigations on the higher protein-split products should be carried on. We have, accordingly, started to analyze the blood of all patients suffering from a toxemia of pregnancy for amino-acids before and after hydrolysis. Our figures are as yet too few to permit of any conclusions.

*Treatment of Eclampsia.*—The chemical and pathologic findings with anesthesia strongly indicate that the eclamptic patient should not be subjected to a general anesthesia. From the results recorded in this paper it seems quite logical to assume that the reduction in maternal mortality in eclampsia incident to the change from radical to conservative treatment[36] is due not to the change in procedure but rather to the fact that a much smaller proportion of patients received general anesthesia.

Our chemical and pathologic findings further indicate that the use of magnesium sulphate to control the convulsions is not warranted. We feel, however, that further work along these lines is required before any sweeping statement can be made.

In the case of morphine and chloral the evidence is of such a character that one may expect beneficial results from their use. Morphia apparently produces no liver lesions, and furthermore, has a definite tendency to raise the carbon dioxide combining power of the blood; chloral, while it seems to give rise to congestion of the liver, does not lead to damage of its cells.

The use of insulin with a protective dose of glucose in severe cases of eclampsia where the carbon dioxide combining power is 30 volumes per cent or lower, and where there is a hyperglycemia, seems quite justifiable from our clinical experience as well as from our experimental studies in animals on the effect of insulin on the various types of hyperglycemia and acidosis. It must be emphasized, however, that as far as we know at present, the only good that insulin may do in such cases is to combat the acidosis; and I feel convinced that certain patients might be saved from a fatal outcome if one could successfully carry them over the marked acidosis so often encountered in eclampsia. It is equally true that we sometimes have a type of eclampsia where acidosis is not a characteristic feature, and yet the patient may succumb. It is

thus clear that our treatment must remain empirical and tentative until we definitely know the cause of eclampsia.

In connection with the acidosis and hyperglycemia of eclampsia, it is interesting to bear in mind the work of Mann[37] and his associates on the physiology of the liver. The blood picture of eclampsia certainly reminds one of the changes produced by removal of the liver, especially when one considers the high uric acid content of the blood together with the changes in blood sugar, lactic acid, and hydrogen-ion concentration. That glucose therapy may be of value in certain types of disturbed liver function seems quite rational. Mann suggests, for example, that glucose may be of value where the supply of glycogen in the liver is depleted, following the marked hyperglycemia of ether anesthesia.[2]

It follows logically that in certain of the toxemias of pregnancy associated with marked disturbance in liver function, as is evidenced by pathologic lesions in that organ, the use of glucose may be of value, and there is clinical evidence to show that this contention may be correct. Duncan and Harding,[38] Titus[39] and others have advocated the use of glucose in the vomiting of pregnancy. It should be stated that in this clinic we have tried the glucose, as well as the insulin and glucose therapy for pernicious vomiting of pregnancy, and that while each of these treatments is of decided value in certain patients, we nevertheless encounter others in whom all attempts at treatment seem futile. This is not to be wondered at when we consider how little is as yet known concerning the etiology of the condition. Here, just as in eclampsia, our treatment must remain empirical and tentative until the true cause of the disease is discovered.

The results of our experimental work on general and local anesthesia lead to the conclusion that in certain of the severe types of eclampsia it may be advisable to end the pregnancy under local or spinal anesthesia, but as yet we have no clinical evidence in support of such a contention. In two control experiments on dogs, novocaine, or procaine, was injected subcutaneously in amounts greater than are ordinarily used in practice, and the blood chemistry studied. Twenty cubic centimeters of a 2 per cent solution of procaine were injected subcutaneously and produced no change in the blood constituents during a two hour period of observation in which samples of blood were withdrawn and analyzed at fifteen minute intervals.

As an analysis of the results obtained in the conservative treatment of ante- and intrapartum eclampsia up to March 31, 1926,[42] shows that the mortality was 1.9 per cent in the mild cases according to Eden's classification,[40] and 21 per cent in the severe cases, it appears that the treatment of the former by the Stroganoff technic[41] gives a very satisfactory result; while in the case of the severe type, improvement is

urgently demanded. For this reason, we have tentatively concluded to continue to treat the former by the Stroganoff method but to ascertain what results will follow the prompt delivery of severe cases under spinal anesthesia. When, however, the patient seems to be in danger from a profound acidosis as evidenced by a fall of the $CO_2$-combining power to 30 volumes per cent or less, we shall continue to use insulin and glucose which our experience up to this time indicates is of value in combating that condition.

## CONCLUSIONS

1. Ether, chloroform, nitrous oxide, and ethylene produce changes in the blood constituents very similar to those seen in eclampsia.

2. These general anesthetics also produce pronounced liver lesions as well as less marked changes in the kidneys.

3. The use of these general anesthetics in the treatment of eclampsia seems open to objection.

4. Blood studies on anoxemia and eclampsia suggest that in the latter condition deficient oxidation may play a part.

5. Peptone, albumose, and histamine produce a blood picture suggesting an anhydremia. The evidence so far adduced, both chemical and pathologic, makes it improbable that any one of them is to be regarded as an etiologic factor in the causation of eclampsia.

6. Peptone, albumose, and histamine produce degenerative liver lesions similar to those associated with vomiting of pregnancy, but as yet we hesitate to assume that they play any etiologic rôle in its production.

7. The fact that morphine raises the $CO_2$-combining power of the blood and does not damage the liver, affords justification for continuing its use in the treatment of eclampsia.

8. The chemical and pathologic findings with magnesium sulphate speak against its use in eclampsia; but further work is necessary before a definite conclusion can be reached.

9. The use of glucose, as well as that of insulin and glucose, seems to be of value in certain cases of vomiting of pregnancy and eclampsia, but not in all.

10. In our experience, a modified Stroganoff technic, has led to a marked reduction in the mortality in mild cases of eclampsia.

11. The treatment of severe cases of eclampsia is not yet satisfactory and it is a question whether prompt delivery under spinal anesthesia may not give better results than we have heretofore obtained.

I am indebted to Dr. J. Whitridge Williams for his assistance in the histologic part of this work, and I am also grateful to Drs. E. K. Marshall, Jr., and E. M. K. Geiling for their many helpful suggestions. The credit for most of the lactic acid determinations belongs to Dr. A. H. Roderet to whom I am also grateful for assistance in many of the experiments.

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# HEMOSTASIS IN VAGINAL HYSTERECTOMY FOR PROCIDENTIA*

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THE female pelvic organs may reasonably be expected to be complicated at times by various pathologic conditions which modify somewhat the indications and the usual technic for vaginal hysterectomy. One of these is a relaxation of the pelvic fascia which permits the uterus to descend and is usually associated with rectocele and cystocele. Histologic study of the fascia endopelvina demonstrates that it is developed by mesothelial condensations around the ureters and the pelvic nerves and blood vessels. Therefore, it seems reasonable to suggest that if the uterus is to be removed and the pelvic fascia utilized to support the bladder and rectum in cases of uterine prolapse, due consideration should be given to the pelvic veins and the technic for vaginal hysterectomy should be developed upon the principle of careful hemostasis.

Before describing such technic, it is essential to discuss briefly the reasons for removing the uterus in conditions of prolapse and to mention some of the complications that may be encountered. In cases of procidentia, it is frequently difficult to decide when to remove the uterus, and, if the uterus is to be removed, to weigh the advantages of abdominal over vaginal hysterectomy. It is also difficult to devise a method to suspend satisfactorily the vaginal vault after vaginal hysterectomy or to determine the limitations for justifiable pelvic dissection. The factors upon which surgical judgment should be based to make wisely these decisions vary with the individual patient and the experience of the operator. These self-evident statements are made because it is well known that vaginal hysterectomy is not always indicated, because cures of procidentia can at times be obtained by simple anterior colporrhaphy, perineorrhaphy and abdominal fixation of the uterus or by some modification of the interposition operation. Unfortunately, however, as with all hernias, there is invariably some percentage of recurrence after operation and this fear of recurrence is the primary reason for the great variety of procedures advocated for the cure of procidentia.

To minimize the danger of recurrence, some gynecologists recommend that the uterus be removed and the pelvic fascia be utilized to

*Presented at the Fifty-first Annual Meeting of the American Gynecological Society, Stockbridge, Mass., May 20, 21, and 22, 1926.

support the bladder, the vaginal vault and the rectum. To do this properly it is necessary to dissect rather extensively the vascular pelvic fascias and in so doing there is danger of operative and postoperative hemorrhage unless particular attention is given to complete hemostasis.

During the past fourteen years, 603 patients suffering with 1122 varieties of pelvic prolapse such as cystocele, rectocele and procidentia of the uterus have been operated upon in the Women's Clinic of the Stanford University School of Medicine. In an effort to determine the best way to permanently cure the largest number of these patients, with the least operative risk and the shortest period of hospital convalescence, various procedures have been utilized. Many patients have been observed for a long period of time in the follow-up clinic and several papers have been published which give the facts as we have observed them in regard to the percentage of recurrence after various methods of operation and the operative and postoperative complications experienced.[1-2]

In regard to recurrence, a recent study of 184 patients with pelvic prolapse showed that later in the clinic there was noted some recurrence in 21 patients or 11.4 per cent. Nineteen of the 21 patients had recurrent rectocele, 9 patients had recurrent cystocele and 2 patients had recurrent prolapse of the uterus. Sixty-two of the 184 patients in this series had the uterus removed either because they were past the menopause or because the uterus was pathologic which accounts somewhat, but not entirely, for the low incidence of recurrent prolapse of the uterus. Of the two patients having recurrent prolapse of the uterus, one followed an interposition operation and one followed a subtotal vaginal hysterectomy.

The operative and postoperative complications associated with hemorrhage have given the greatest concern and have influenced in a marked way, the development of the operative technic in the clinic. Of the 603 patients operated upon, one had severe postoperative hemorrhage following vaginal hysterectomy, two patients developed pelvic hematoma, one after a Jellett operation and one after a subtotal vaginal hysterectomy and two patients died with the symptoms of postoperative shock, both after subtotal vaginal hysterectomy.

These experiences have caused us to give up the subtotal vaginal hysterectomy[3] because we could not perfect a satisfactory technic to control hemorrhage without occasionally experiencing some degree of cervix sloughing which increased the incidence of recurrence. Since 1921, we have not done the Jellett operation[4] because with the extensive dissection involved, more difficulties were encountered with hemostasis than in cases where the uterus was completely removed. We reluctantly gave up the Goffe operation,[5] not because of hemorrhage,

but because one patient after operation developed a ureterovaginal-vesicovaginal fistula which was most difficult to close.

With the development of the present technic for hemostasis we have

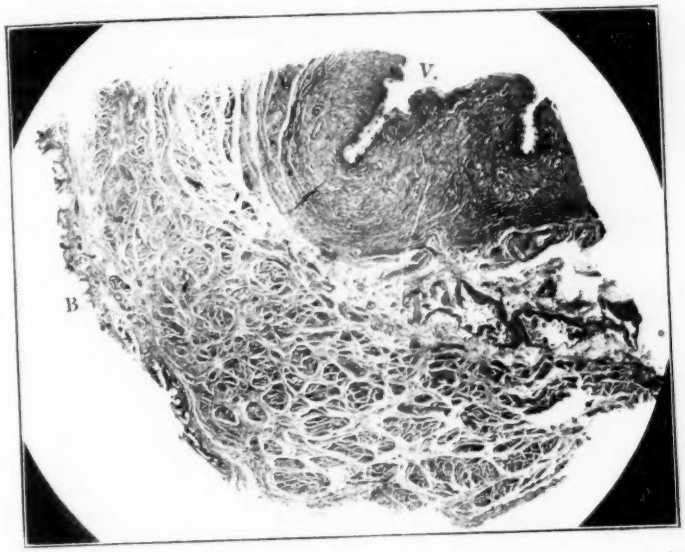


Fig. 1.—Section of tissue between bladder and anterior wall of vagina, (x4). *B*, bladder. *V*, vagina.

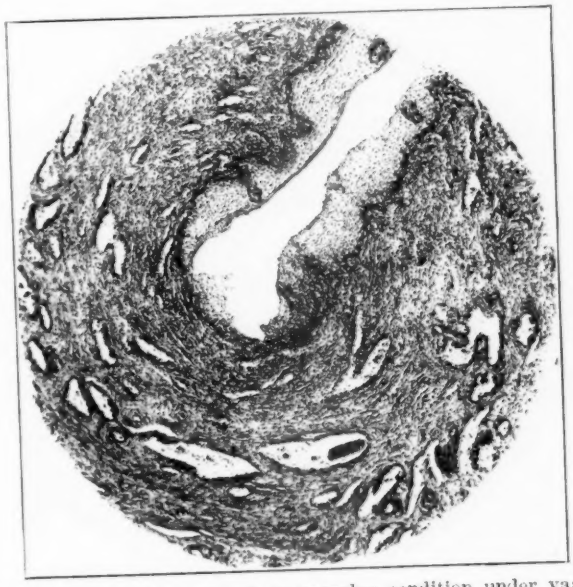

Fig. 2.—Vesicovaginal fascia to show vascular condition under vaginal wall.

come to rely more and more upon complete vaginal hysterectomy for the cure of procidentia in women near or past the menopause.

In 1919, I presented to this Society a study of frozen sections of

the pelvis,[3] the photographs of which showed very clearly the fascia endopelvina. Since that time, further anatomic study of the pelvic fascia has been made. Some of the points brought out by these ana-

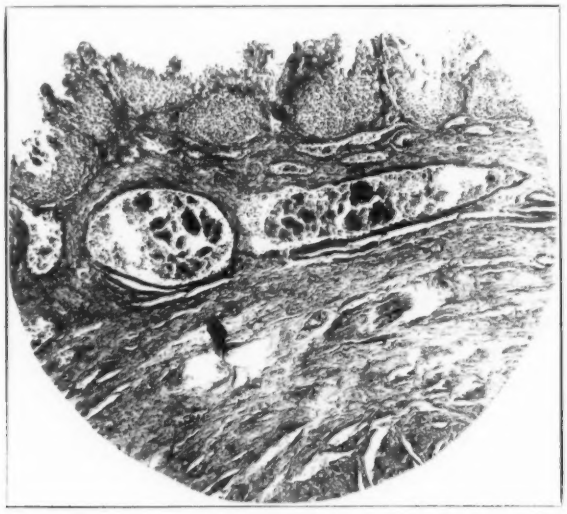

Fig. 3.—Vesicovaginal fascia to show vascular condition under bladder wall.

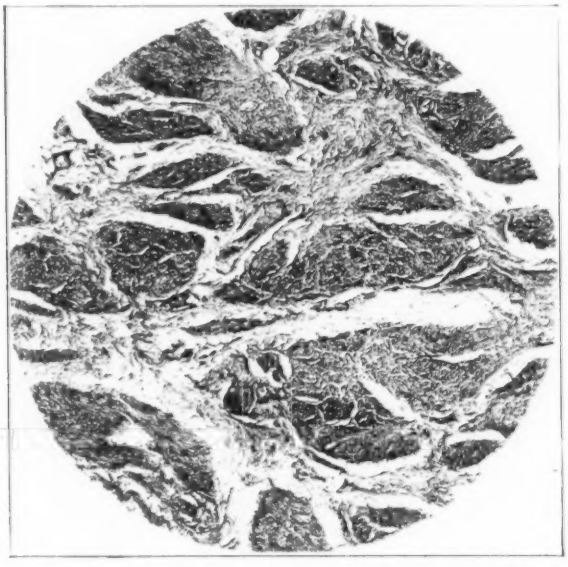

Fig. 4.—Vesicovaginal fascia. Intermediate portion.

tomic dissections have been utilized not only in the technic for the support of the bladder in cases of cystocele, but for the control of hemorrhage in vaginal hysterectomy. Fig. 1 shows a section of the tissue between the bladder and anterior wall of the vagina. Figs. 2,

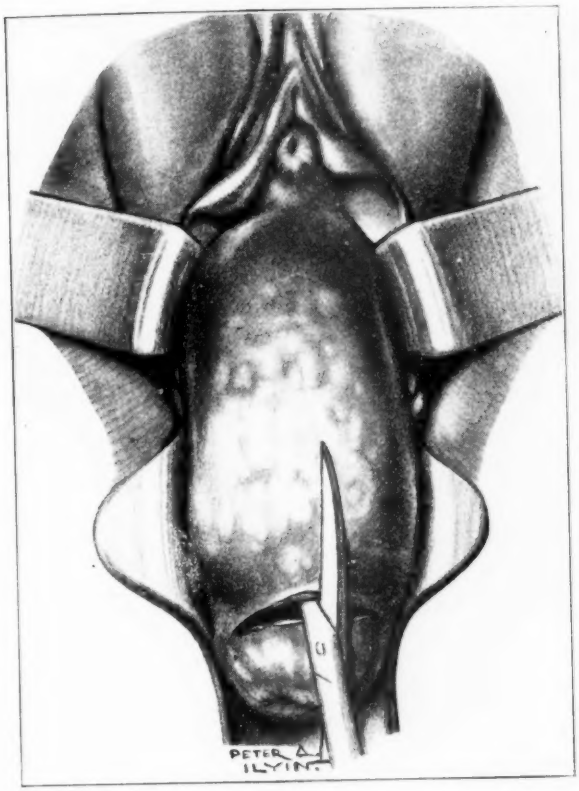


Fig. 5.—Separation of vaginal wall and vesicovaginal fascia from the bladder.

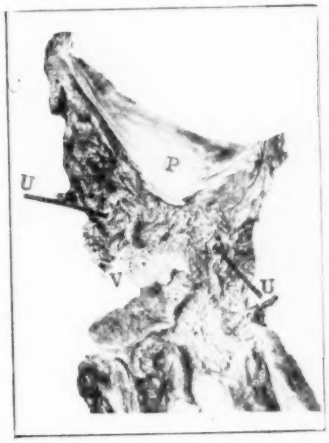


Fig. 6.—Posterior view to show relation of ureters to vesicouterine ligaments. *P*, Peritoneum of Douglas' pouch. *U*, ureters. *V*, vagina at cervix.

3, and 4 show the vascular condition of this tissue just under the vaginal wall and under the bladder wall with a thick muscular non-vascular layer lying between. The practical utilization of this vesico-

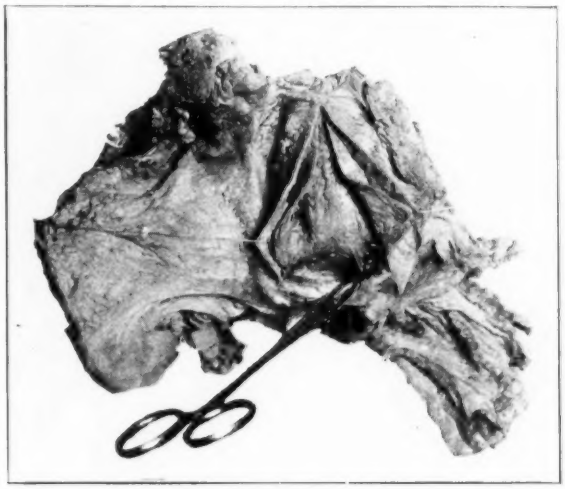

Fig. 7.—Anatomical dissection which shows anterior vaginal wall with dissected vesicovaginal fascia and bladder. Clamp has been placed on the left vesicouterine ligament.

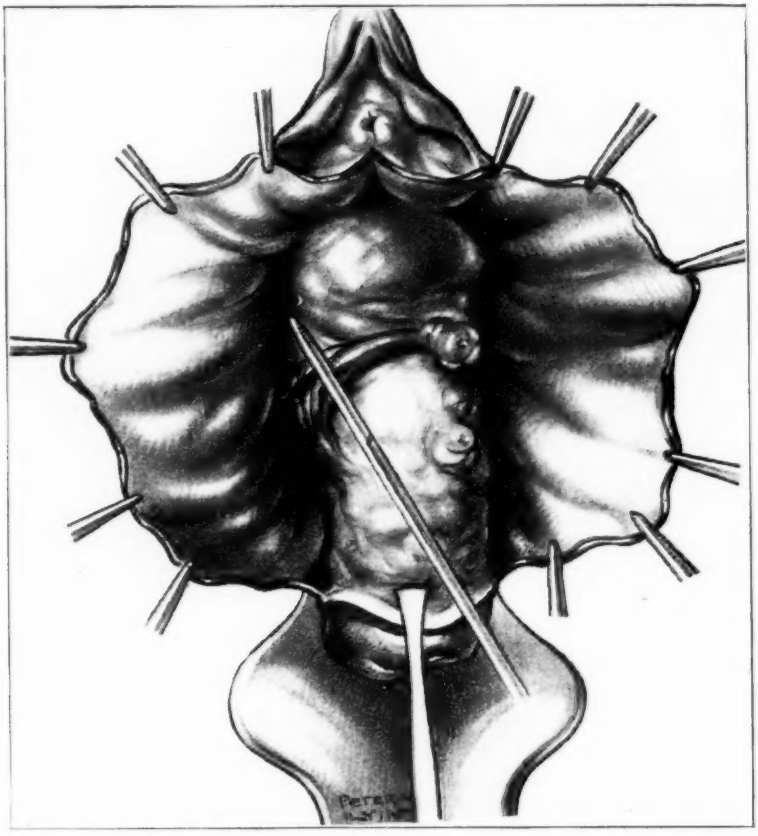

Fig. 8.—Ligation of left vesicouterine ligament. Hemostat placed on right vesicouterine ligament, previous to section.

vaginal fascia in the repair of cystocele as advocated by the late Dr. J. Craig Neel,[6] of San Francisco and by Dr. R. M. Rawls of New York,[7] has been made for the past ten years with fairly satisfactory results in regard to the incidence of cystocele recurrence. In separating this fascia from the bladder as shown in Fig. 5, the deep vascular layer should not be removed as it forms an important part of the bladder wall. The vesical plexus of veins lying in this part of the

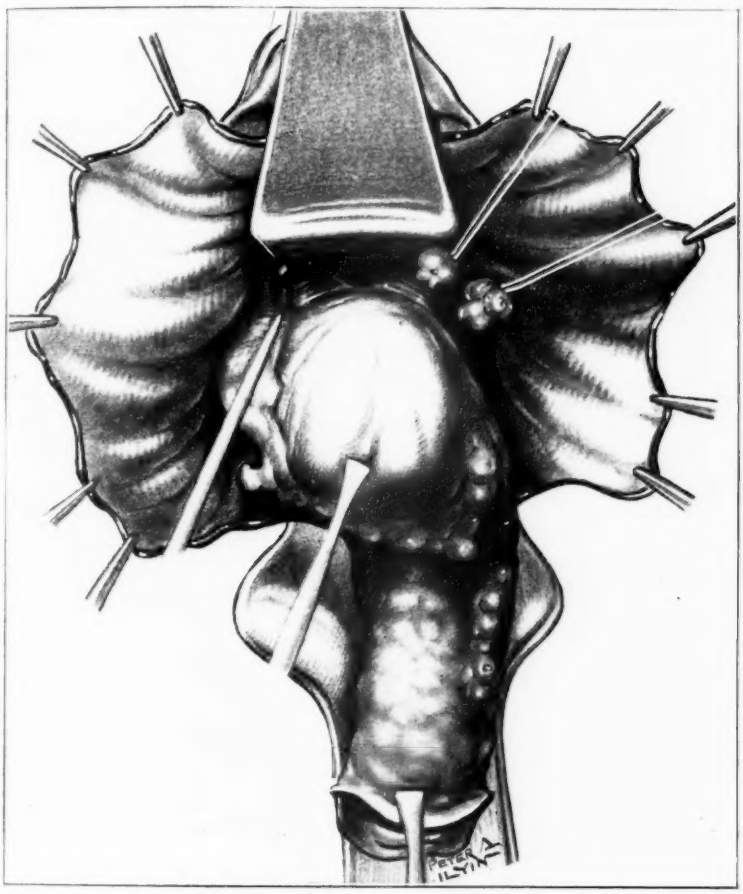

Fig. 9.—Delivery of fundus of uterus under Doyen clamp which protects bladder, to show separate ligation of round ligaments and broad ligaments.

fascia with the surrounding mesothelial condensations passes down the anterior wall of the bladder to form, on either side, the thick vesicouterine ligaments as the vessels pass to empty into the uterine veins. This fascia comes in quite close contact to the ureter as shown in Fig. 6 but can easily be clamped and ligated without injury to the ureter or injury to the bladder wall if the clamp is placed close to the uterus as shown in Fig. 7. With separate ligation of the vesico-

uterine ligaments not only can the bladder be more completely supported but the control of the venous oozing is perfect. By freeing the bladder completely from the anterior vaginal wall and from the midportion of the anterior uterine wall, the bladder can be lifted from the uterus producing tension on the two vesicouterine ligaments. These ligaments can then be safely clamped close to the uterus with a small straight hemostat if reasonable care is taken that the ureter

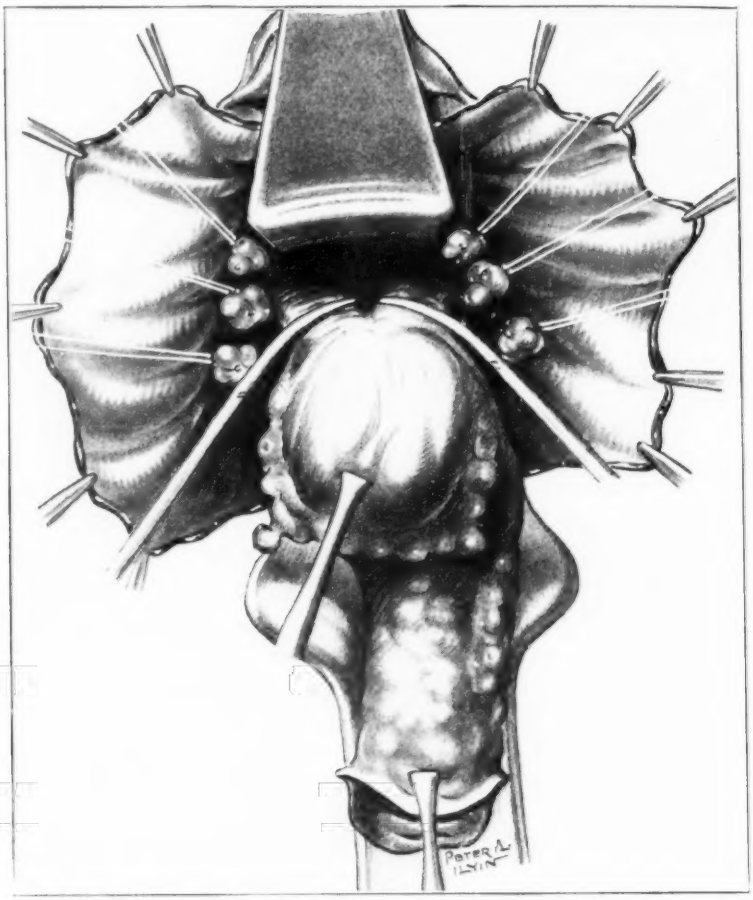

Fig. 10.—Separate ligation of sacrouterine ligaments.

is not in the clamp. The ligament can be cut close to the uterus and sutured on the bladder side with a ligature of small chromic gut around the clamp (Fig. 8). This technic has been used for over four years without injury to the bladder or ureter and with satisfactory control of operative and postoperative hemorrhage. After severing these ligaments, arterial hemorrhage is controlled by exposing and ligating the uterine arteries. The double ligatures on the uterine arteries are cut short but no attempt is made to utilize the base of

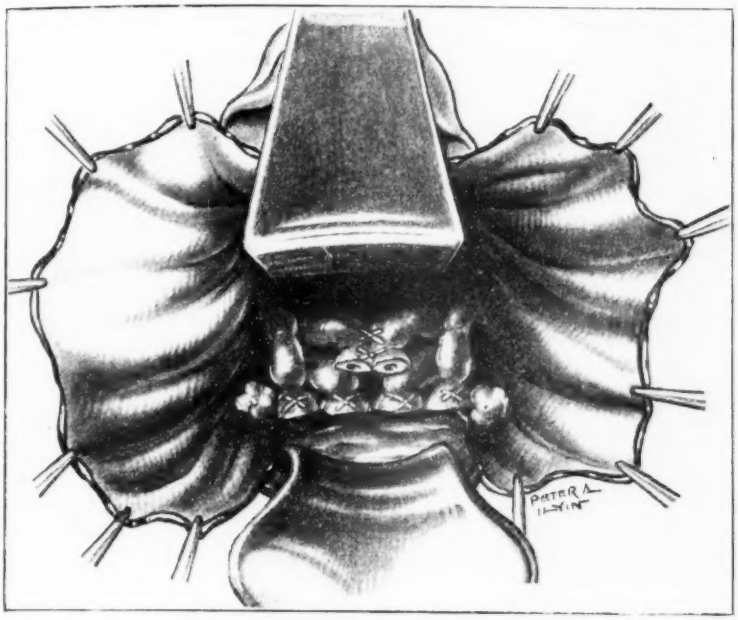

Fig. 11.—Round ligaments and sacrouterine ligaments sutured to posterior vaginal wall. Broad ligaments sutured under Doyen clamp.

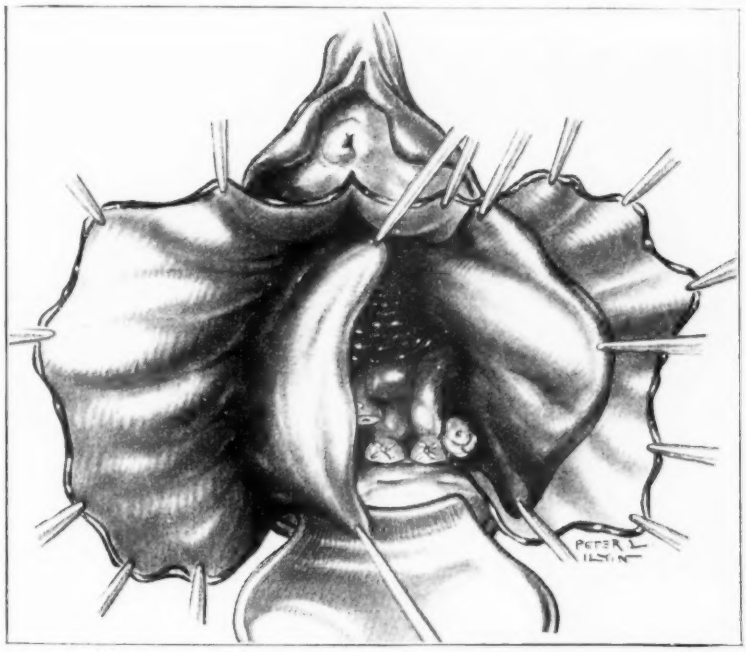

Fig. 12.—Vesicovaginal fascia has been separated from the vaginal wall to near the white line. Overlapping this fascia and suturing lower edge to vaginal wall supports the bladder.

the broad ligaments for the support of the vaginal vault as this would increase the danger of postoperative hemorrhage.

After opening the peritoneal cavity, under the bladder, the bladder can be protected with a wide Doyen retractor and the omentum and intestines packed off. This permits of delivery of the fundus of the uterus and separate ligation of the round ligaments and the adnexal connections (Fig. 9). By traction on the cervix and uterine fundus, the sacrouterine ligaments become taut and can readily be clamped, ligated and cut (Fig. 10). The uterus is then removed by section of the posterior vaginal wall. After suturing the sacrouterine ligaments and the round ligaments to the severed posterior vaginal wall, the stumps of the broad ligaments are brought together just under the

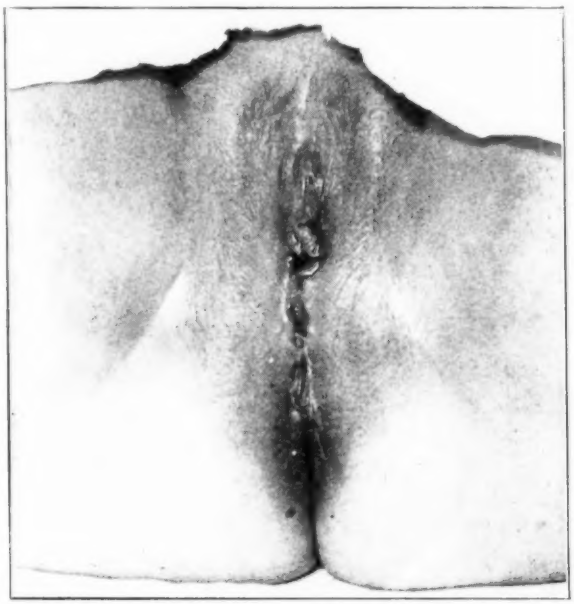

Fig. 13.—Shows condition of patient on leaving hospital fourteen days after operation.

junction of the urethra to the bladder (Fig. 11). Removing the retractor and the abdominal pack, a circular suture through the cut edges of the vagina, the round ligaments and the broad ligaments, closes the vaginal opening. If the vesicovaginal fascia has been freely dissected from the bladder and from the vaginal mucosa, it will usually be found to be quite thick. Overlapping this fascia (Fig. 12), suturing it to the edge of the vaginal vault under the bladder and closing the mucosa of the anterior vaginal wall with interrupted sutures, completes the operation. Almost always a proctopexy[2] and a perineorrhaphy is needed following which a light vaginal pack is usually placed. Fig. 13 shows the condition of a patient on leaving the hospital.

## CONCLUSIONS

In a series of 603 patients suffering with various conditions of pelvic prolapse, 90 have been treated by complete vaginal hysterectomy. Experiences with this operation have demonstrated the need for wide dissection of the pelvic fascia to close the hernial opening and minimize the danger of recurrence. As the pelvic fascia is developed around the ureters, the nerves, and especially the pelvic vessels, special technic is necessary to guard against postoperative hemorrhage. The particular point in such technic as illustrated in this paper has to do with the separate ligation and section of the vesicouterine ligaments.

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[3]Spalding, Alfred Baker: Tr. Am. Gynec. Soc., 1919.
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[6]Neel, J. Craig: Calif. State Jour. Med., April, 1917, xv, 121.
[7]Rawls, Reginald M.: Am. Jour. Obst., March, 1918, lxxvii, 359.

(*For discussion see page* 765.)

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# THE TREATMENT OF GRANULOMA INGUINALE WITH TARTAR EMETIC*

By John A. McGlinn, M.D., Philadelphia, Pa.

SOME years ago I reported four cases of tuberculosis of the vulva, occurring on my service at the Philadelphia General Hospital. In the light of our present knowledge, we know that the cases were not tuberculosis, but granuloma. For fully fifty years this disease has been endemic in the hospital and was variously diagnosed as lupus or syphilis. It was not until 1921 when Randall, Small and Beck, becoming interested in the study of granuloma, found seven cases of the disease in the wards of the hospital, that the entity of the condition was recognized in Philadelphia. Since their studies the disease is regularly diagnosed and there is never a time when we do not have one or more cases in the hospital.

The true nature of the disease has been recognized for a number of years but, inasmuch as the early papers were published by South American physicians and as the disease is almost always found in negroes, it was regarded as a tropical disease and little or no attention was paid to it in the north temperate zone. Following the papers of Symmens and Frost in 1920 and Randall *et al* in 1921, interest was aroused in the subject with the result that cases have been recognized and reported from every section of the United States.

*Read at the Fifty-first Annual Meeting of the American Gynecological Society, Stockbridge, Mass., May 20, 21 and 22, 1926.

All of our cases with two exceptions have occurred in the negro. One of our white cases was in a young married woman, age twenty-five, whose husband was free of venereal disease and whose personal habits of cleanliness were excellent. Before consulting us she had been a patient in two of our largest hospitals and in both the condition was diagnosed as inopererable cancer of the vulva and treatment by x-ray instituted. The case was, clinically, granuloma and biopsy and bacteriologic studies corroborated the clinical diagnosis . She refused treatment, left the hospital, moved from her former address and could not be traced.

The lesion starts usually with a small noninflammatory papule on the vulva. The papule ruptures and exudes a purulent fluid. Unlike

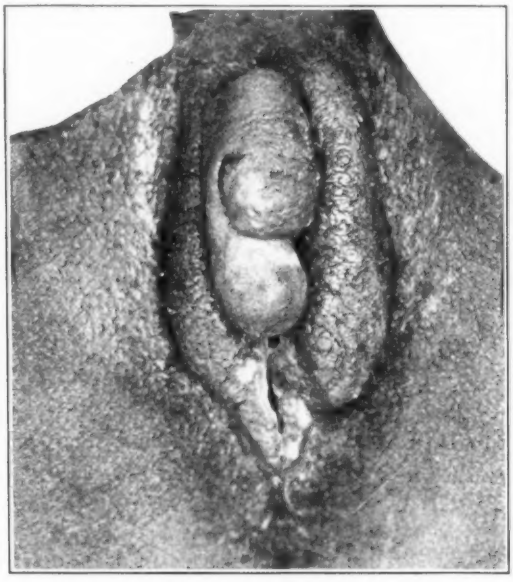

Fig. 1.—Early case of granuloma, showing an edema of the vulva.

most papules that rupture, healing does not take place but on the contrary spreads by slow proliferation. In the early stage, while it does not resemble a phagedenic ulcer, it is often mistaken for one and the usual escharotic treatment instituted. The typical lesion is that of exuberant granulation tissue, which is soft in structure and red in color. There is usually destruction in the center of the growth but the edges are exuberant and usually overlap the healthy skin edges. The surface is covered with a scanty mucoid exudate which, while nonoffensive, has a peculiar odor. I have frequently been able to anticipate the diagnosis of the case from the odor before seeing the lesion. When the exudate is removed a clean, red, healthy-looking granulating area is revealed. The advanced cases show large

granulating areas, with here and there cicatricial tissue and on the edges papules of new growths. It was this picture of beginning new growths, advanced growths and areas of healing that caused it to be confused with lupus.

The usual site of the disease is the labia majora but in the advanced cases the entire vulva and groin are involved. In not a few cases extension into the vagina has been observed and in one of our cases the rectovaginal septum was destroyed. Lymphatic circulation is interfered with and edema of the vulva is frequently observed.

Except for the presence of the growth there are few subjective symptoms. The lesion is painless and the usual symptoms and blood findings of infections are absent.

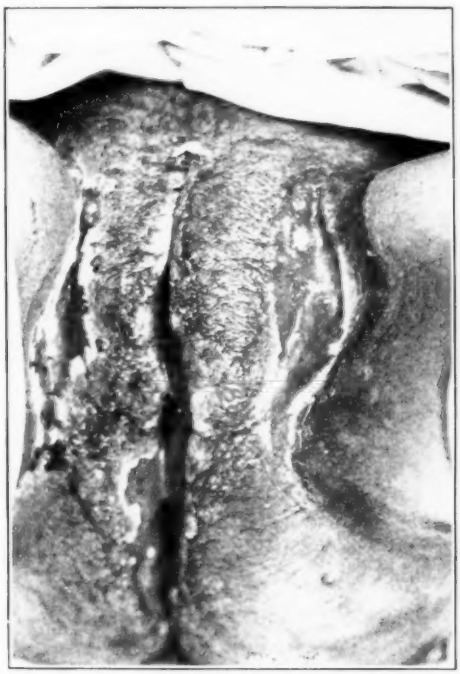

Fig. 2.—Advanced case of granuloma showing exudate.

Its insensitiveness makes it possible for the lesion to become an interesting complication of pregnancy. The presence of pregnancy in a well advanced case of granuloma proves the futility of attempting to inhibit the indulgence of natural desires.

Five pregnancies have occurred in three of our cases and we were unable to note any favorable or unfavorable effect upon the lesions. In all these cases the disease was far advanced, involving not only the entire vulva but the posterior wall of the vagina. Our most noted case, A. W., has had three pregnancies, the last in spite of the fact that she had a large rectovaginal opening with a profuse discharge

of feces into the vagina and over the vulva. In these three cases it was felt that vaginal delivery was contraindicated on account of the danger of infection and severe traumatism. Five cesarean sections were done with no fetal and one maternal death from pneumonia and paroxysmal tachycardia. One patient had three cesarean sections; one done on the service of Dr. Edward Schumann and was reported by him before a joint meeting of the New York and Philadelphia Obstetrical Societies several years ago.

The more or less definite clinical picture of granuloma makes the diagnosis comparatively easy, even without the aid of bacteriologic and histologic studies. In our cases, however, we make routine smears and biopsies and submit them to the laboratory for study. Little has

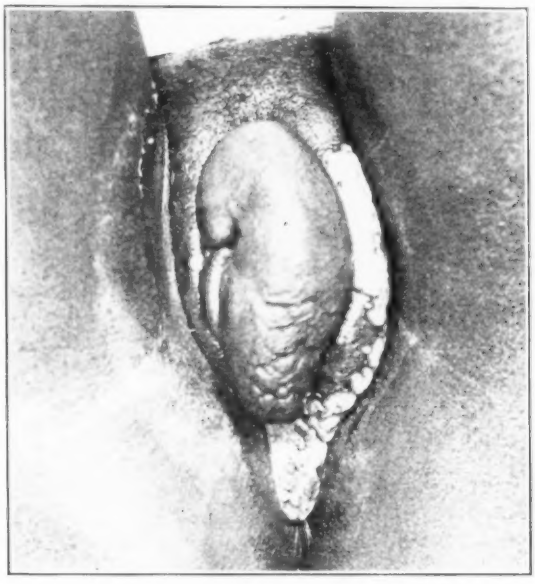

Fig. 3.—Moderately advanced case of granuloma showing exudate and edema of the vulva.

been added to our knowledge of the bacteriology and histology of granuloma since J. C. Small's studies made in our laboratories in 1921 and 1922, and whose findings will be freely quoted in this paper. As stated previously, mistaken diagnoses were always made prior to 1921. The most common error in diagnosis was to consider granuloma lupus or syphilis. Many other mistaken diagnoses were made, such as chancroidal sores, cancer, condylomata accumminata and condylomata lata. As might be expected many cases present a positive Wassermann and in former days were considered syphilis. As we have become familiar with the clinical picture of granuloma mistaken diagnoses are rarely made.

Direct smears from the lesion, made with a stiff wire loop, stained

either by the Wright or Giemsa method show the characteristic encapsulated bacillus first described by Donovan. When well stained the organisms appear as small, rounded, pink bodies with a dark blue coccoid body in the center; or more frequently as oval pink bodies with a blue bacillary or diplococcoid body occupying the longitudinal axis. The pink outer zone is a wide capsule. The dark blue central bodies represent metachromatic granules within the body proper. The organisms are found within the cytoplasm of large mononuclear cells. Bipolar staining may be observed at times in these nests of organisms. Encapsulated organisms may appear free in the cellular detritus in the neighborhood of disintegrating mononuclear cells.

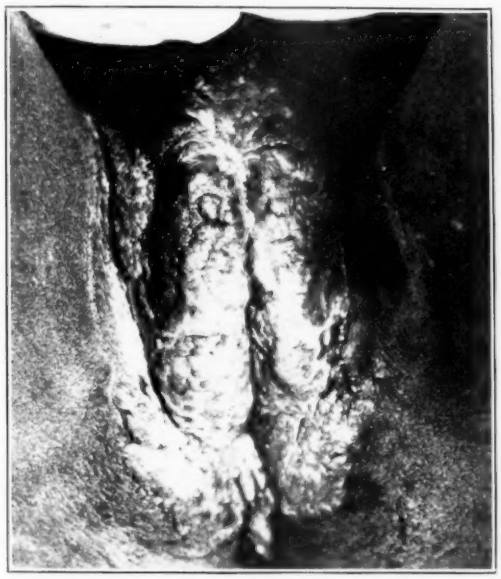

Fig. 4.—Advanced case of granuloma with rectovaginal fistula. Three pregnancies, all terminated by cesarean section.

Smears are but little contaminated by organisms usually present in other vulva lesions.

Small has made a number of cultural studies of the organisms and has reported them in *The Journal of Infectious Diseases*, Vol. 32, June, 1923, and in *Surgery, Gynecology and Obstetrics*, June, 1921. Animal and human inoculations have failed to produce the characteristic lesions. The Donovan bodies are universally found in fresh smears in untreated cases and disappear entirely after two or three treatments with tartar emetic.

Biopsy sections when stained show a superficial cellular area on a base of dense hyaline connective tissue. The cellular area is composed of young connective tissue, relatively small in amount, many endothelial leucocytes, and a smaller number of polymorphonuclear

neutraphiles. Many leucocytes are also present and an occasional eosinophile. At the margin of the granulation the squamous epithelium of the skin is partially destroyed and replaced. Further out this merges into normal skin, under which, however, the subcutaneous tissues are infiltrated by round cells.

There is a proliferation of squamous epithelium near the edges of the granuloma where finger-like projections extend into the deeper tissues resembling to some extent squamous cell carcinoma. After treatment with antimony dense fibrous tissue replaces, to a large extent, the granulations.

Tartar emetic is a specific in the treatment of this disease. Local treatment and excision are of no value and x-ray, while of some bene-

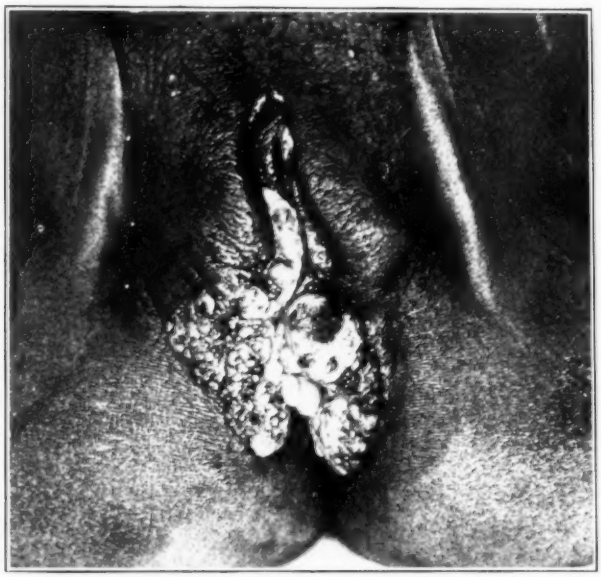

Fig. 5.—Granuloma involving anal region.

fit, cannot be used sufficiently long to effect a cure. We rely entirely on the intravenous use of tartar emetic and local cleanliness. While this drug is a specific it has several disadvantages, which makes its use impossible for a long enough period, in many cases, to effect a permanent cure. One-tenth gram of the drug is dissolved in 10 c.c. of sterile salt solution and given intravenously at weekly intervals in ambulant cases and every other day in hospital cases. The action of the drug is prompt; beginning healing can be observed within forty-eight hours after the first injection. The number of injections required to completely heal the lesion depends on its extent and location. Lesions in the female heal less rapidly than those in the male.

One disadvantage of antimony injections is the tendency to obliter-

ate the veins. For this reason we start the injections into the veins on the back of the hand and then go higher in the arms as the veins below become obliterated. In a few of our cases we have been obliged

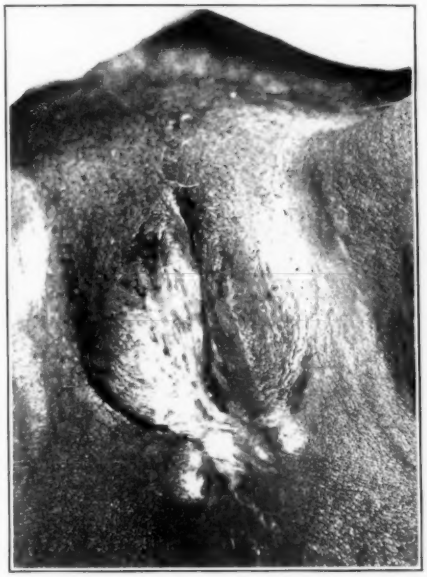

Fig. 6.—Granuloma showing appearance of the lesion after treatment with tartar emetic.

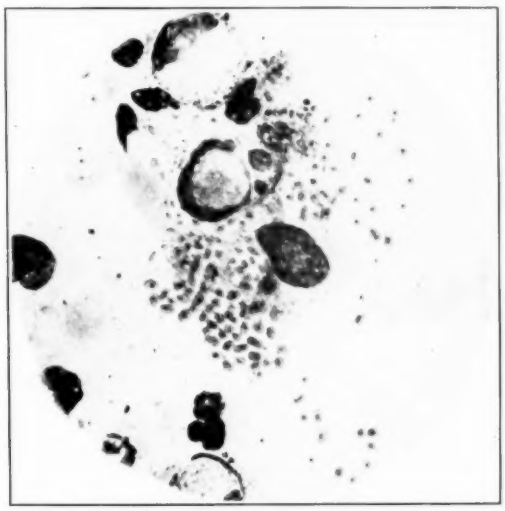

Fig. 7.—Photomicrograph of smear taken from granuloma showing Donovan's bodies (after Small).

to discontinue treatment before complete healing had occurred because of obliteration of all available veins. As far as I know no satisfactory intramuscular preparation has been perfected.

The second objection to antimony is the severe rheumatoid joint pains which follow within twenty-four hours after the injection. At times these pains are so severe that patients refuse to continue treatment. A case should not be considered permanently cured just because complete healing has taken place. These cases have a tendency to recur and should have several courses of treatment after the lesion has completely healed. It is practically impossible to keep these patients in the hospital once their lesions are healed and on their discharge they are instructed to report to the clinic for further treatment. This they seldom do until they have a recurrence, when they are again admitted to the wards. They suffer so much from the treatments that they will not return for the injections as long as they are well. Except for these rheumatoid pains we have not observed any serious effects from the use of tartar emetic. Randall observed several cases of kidney insufficiency following its use and realizing the toxic nature of the drug sought an antimonial compound of lesser toxicity. He obtained from Professor J. J. Abel of the Johns Hopkins University two antimonials, sodium antimony thioglycollate and a new synthetic composition the triamide of thioglycollic acid. Cases treated with these two preparations healed as promptly as others in which tartar emetic was used and there was an entire absence of toxic symptoms and rheumatoid pains. Randall has had these two compounds prepared by a prominent pharmaceutical house and is making a study of their therapeutic value.

In conclusion it is to be remembered that inguinal granuloma is endemic in northern latitudes and any vulvar lesions which resist ordinary treatment should be suspected of being granuloma. Antimony is a specific for this disease. The lesions have a tendency to recur and a series of ten intravenous injections should be given after complete healing has been secured.

1530 LOCUST STREET.

(*For discussion see page* 762.)

# SUPPRESSION OF URINE IN CONNECTION WITH PREGNANCY*

By John C. Hirst, 2nd, M.D., Philadelphia, Pa.

*(From the Department of Obstetrics, Hospital of the University of Pennsylvania)*

THIS report includes three types of anuria complicating pregnancy and the puerperal state: (1) complete suppression due to kidney degeneration, sometimes spoken of as "idiopathic anuria"; (2) suppression due to urinary calculus, and (3) that due to ureteral edema or pressure.

The first example is that of a woman who lived for nine days with complete anuria following the birth of a stillborn fetus at seven and one-half months.

Case 1.—Mrs. E. R., aged thirty, was admitted to the University Maternity April 27, two days after an easy spontaneous stillbirth. There is no accurate information concerning the cause of the fetal death, or the duration of the retention *in utero* of the dead baby, but there are the facts that the patient had been well during this pregnancy, had never shown evidence of kidney impairment or of any other serious illness beforehand, and that she had previously given birth to one healthy child.

The bladder had been found empty before and after sweats, enteroclysis, and purgation at home. Our examination on admission showed a well-appearing woman, with normal temperature, pulse, and respiration, but considerable abdominal distention. The pelvic organs were in average postpartum condition, the bladder containing one dram of ammoniacal urine that showed no albumin and no casts, but many white blood cells. Blood pressure was 110/70.

On April 28 distention was less marked. The patient had no eye symptoms, no headache, no loin pain, and her mind was clear. Cystoscopic examination showed the bladder normal, ureters not obstructed, and no urine at all. Blood pressure, 115/80.

April 29. Blood urea nitrogen 106 mg., and creatinine 7.9 mg. Blood count showed 1,780,000 red cells; 17,400 white cells; hemoglobin, 35 per cent; polynuclears, 78 per cent; lymphocytes 18 per cent, and large mononuclears, 4 per cent.

Wassermann negative. Ophthalmoscopic examination by Dr. de Schweinitz showed only a fine hazy retinal edema. Pyleogram made with 12 per cent sodium iodide solution revealed a normal kidney pelvis and ureter on each side.

May 1. After occasional vomiting, slight headache, dimness of vision, urinary odor, and persistent anuria, bilateral decapsulation of each kidney under gas by Edebohl's method was performed as a last resort. At operation each kidney was found slightly enlarged, pale, mottled, and friable. Immediate recovery was good and the patient passed two ounces of bloody urine in the first twelve hours. The day following, greater pallor of the optic discs was present. Patient was drowsy, blood pressure, 120/80. Amyl nitrite was given for possible spasm of the renal artery, since the usual uremic remedies had failed. The patient developed convulsions and died May 3, having excreted only nine ounces of urine in nine days.

*Read at a meeting of the Philadelphia Obstetrical Society, January 7, 1926.

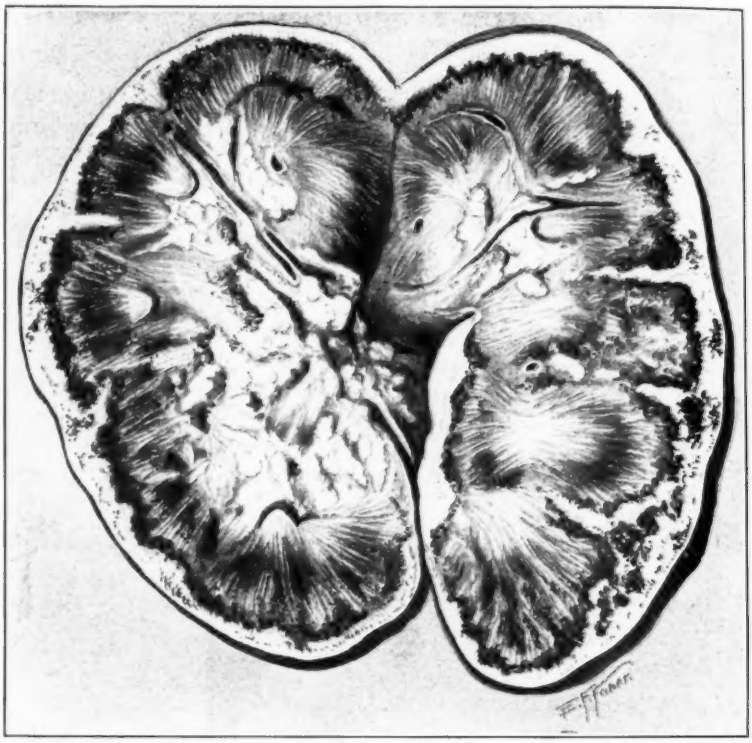

Fig. 1.—Case 1; suppression of urine for nine days from kidney degeneration. (Note cortical necrosis.)

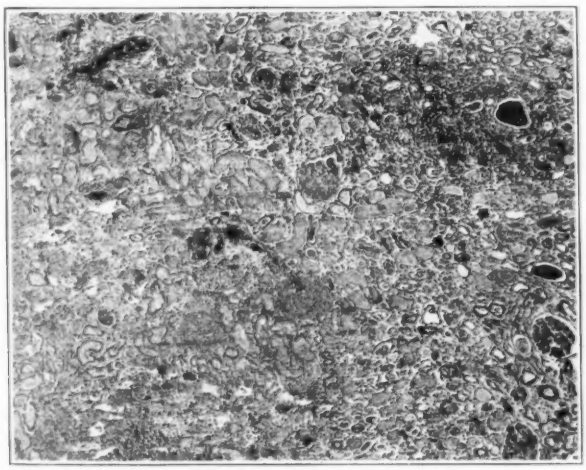

Fig. 2.—Low power section of kidney from Case 1.

The kidneys were removed for examination. No mercury or bacteria in Gram-Weigert section was found. The pathologic report was as follows: "A very definite degenerative nephritis involving the convoluted tubules even to total destruction of nuclei. Many glomeruli show cellular increase, thrombosis and detritus

within the capsule, and a few show fibrin thrombi. Some of the smaller vessels are thrombotic; this kidney seems to present a bacterial and toxic nephritis of recent origin."

The causes of nonobstructive suppression of urine in order of frequency are: First, severe late gestational toxemia with or without underlying nephritis. This is not a complete suppression, but has been treated by decapsulation of the kidneys even recently;[1, 2] B. C. Hirst states that it is due to an exacerbation of a chronic nephritis,[3] and usually is fatal.

Second: Chemical poisoning is usually mercury, occasionally phosphorus. Since mercurochrome has been extensively used for puerperal sepsis, we see relative anuria sometimes. Attention has been called to this feature by A. V. St. George, of Bellevue Hospital,[4] who mentions severe mercury nephritic lesions in eleven autopsies on women treated with mercurochrome for puerperal sepsis.

Third: Degenerative nephritis. This disease appears to be due to a metabolic poison associated with pregnancy. Rolleston has collected eleven cases, showing symmetrical necrosis of the kidney cortex, of which cases most were associated with stillborn premature babies. Berkely and Bonney[5] agree with Rolleston, and state that the condition in no way resembles eclampsia. Boquel[6] and Jardine and Kennedy[7] also believe that a toxic process is responsible as it appears to be in our case. It is possible that products from the dead fetus may injure the kidneys, and if so the early diagnosis of death of the fetus in the uterus becomes more important.

Unfortunately the cultures from the bladder of our patient were lost. Since the course of the disease was afebrile it is probable that bacterial activity was secondary, and that the process in the kidney was mainly a recent toxic degenerative nephritis.

The second example of suppression of urine is that of a patient who presented complete anuria for four days from urinary calculi on one side and ureteral edema on the other, and recovered with surgical treatment.

CASE 2.—Mrs. T. B., aged twenty-four, para ii, past history unimportant. First admission 9/5/25 to 9/17/25, with a complaint of severe right-sided pain, of three days' duration, starting in the lumbar region and radiating to the right groin and leg. Pain was constant except for frequent exacerbations and was accompanied by vomiting. No other complaints except polyuria. Examination showed pregnancy at about the sixth month and considerable tenderness in the right lumbar region and right lower abdomen. Temperature 104°. Urine showed a light cloud of albumin, many pus cells, and a few red cells.

Following routine treatment for pyelitis, cystoscopy was done six days after admission and after three days of normal temperature. An obstruction to the passage of the catheter was found in the right ureter about 5 cm. from the bladder. With the catheter in place an x-ray picture was made, which showed a small shadow lying next to the catheter. The patient being free from fever and symptoms at this time, she signed her release without obtaining further treatment.

Second admission: 10/13/25 to 12/30/25. Readmitted with a history of having had two attacks resembling ureteral colic accompanied by vomiting; she was still suffering with the second attack, which began three days previous to admission. The patient had complete anuria for three days. Temperature, 101.4°; pulse, 120;

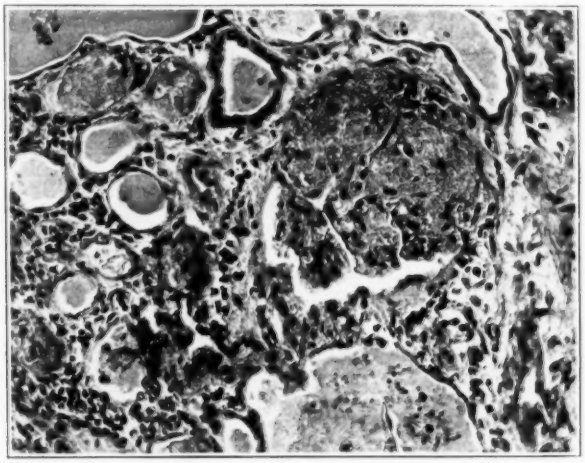

Fig. 3.—High power section of kidney from Case 1.

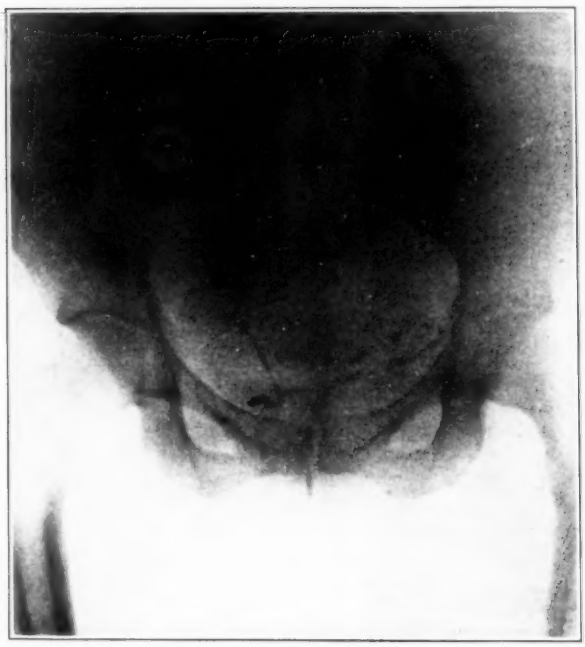

Fig. 4.—Case 2. Pregnant six months. Small stone at tip of catheter.

respiration, 48. Patient was restless and slightly confused. Her skin was dry and hot; her breath was not definitely urinous. General examination was unimportant except for acute deep tenderness in both loins. Pregnancy was then at about the seventh month; head was down, and fetal movements were active. Blood pressure,

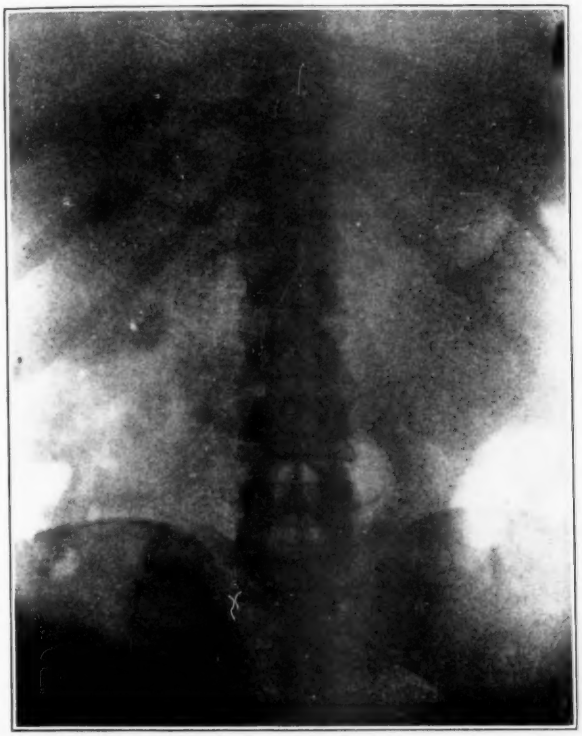

Fig. 5.—Case 2. After delivery. Large stone in right kidney. Enormous left hydronephrosis, from edema of ureter.

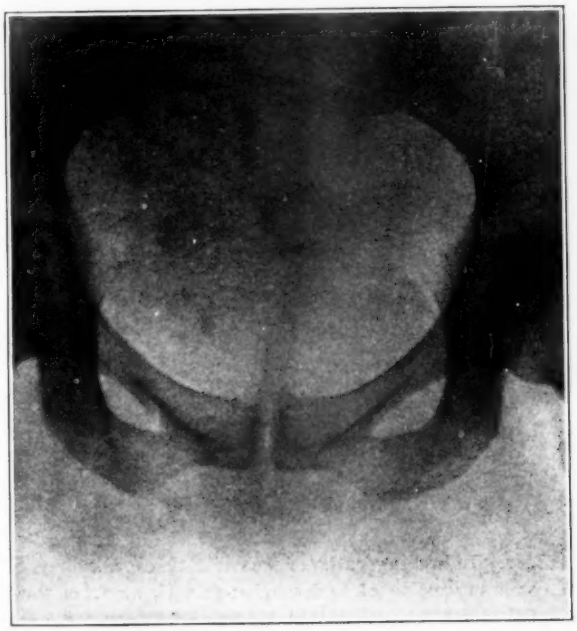

Fig. 6.—Case 2. Three stones in right ureter. (Coincident with stone in right kidney.)

155/90. Leucocyte count, 25,200. Catheterization of bladder yielded no urine. Miscarried the same evening, the child living about three hours.

*Cystoscopy:* Catheterization of the right ureter disclosed an obstruction near the right kidney pelvis, which could be displaced, whereupon purulent urine under pressure gushed from the catheter.

Following this cystoscopy the patient rapidly improved, fever and symptoms subsided, and urinary output increased. Two subsequent cystoscopies were done in the succeeding two weeks, in order to establish and maintain adequate urinary drainage, to complete function studies, and to prepare the patient for lithotomy. In chronologic order, her progress was as follows:

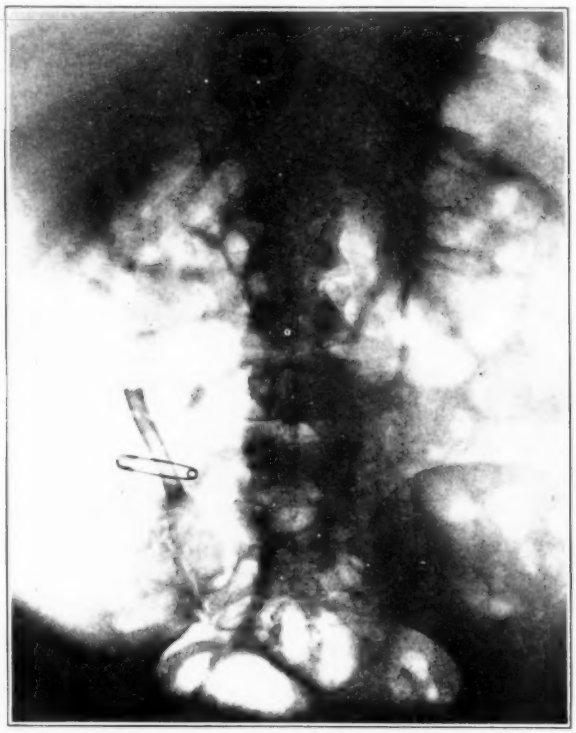

Fig. 7.—Case 2. Stone removed from right kidney.

10/20/25. X-ray shows an enormous left kidney and a large shadow in the right kidney area,—probably renal calculus. In addition there were three small shadows, probably multiple calculi in the lower end of the right ureter.

10/22/25. *Cystoscopy.*—The right ureter did not admit any catheter more than 2 cm. and drained no urine. The left ureter admitted a No. 6 catheter to the kidney pelvis without obstruction, and drained moderately cloudy urine very rapidly. Differential function with indigocarmine appeared at left ureteral orifice in seven minutes; none at right ureteral orifice in fifteen minutes.

10/29/25. *Cystoscopy.*—Right ureteral orifice showed a small grey stone protruding into the bladder. Indwelling catheter was left in each kidney pelvis. Rate of flow on left side was suggestive of hydronephrosis. Urine from the right side was slightly cloudy. Oil was left in the right kidney pelvis. Blood urea nitrogen, 26; uric acid, 4.2.

11/7/25. *Right nephrolithotomy.*—A soft grey stone, about 3 cm. in diameter found in the right kidney pelvis was removed with difficulty, being crushed in the attempt. Lithotomy was performed through Edebohl's paravertebral incision. Wound was packed with plain gauze, and one cigarette drain was inserted.

11/24/25. Patient passed one stone, the size of a pea, by urethra.

12/7/25. Urinary fistula, through lumbar wound. Patient was passing an adequate amount of urine per urethra.

12/11/25. She passed a second stone by urethra.

12/14/25. There was no x-ray evidence of renal, ureteral, or vesical calculus.

12/30/25. Patient discharged in good condition. Urinary fistula closed for past week. Superficial sinus at lower end of lumbar wound.

Final catheterization of ureters showed no obstruction.

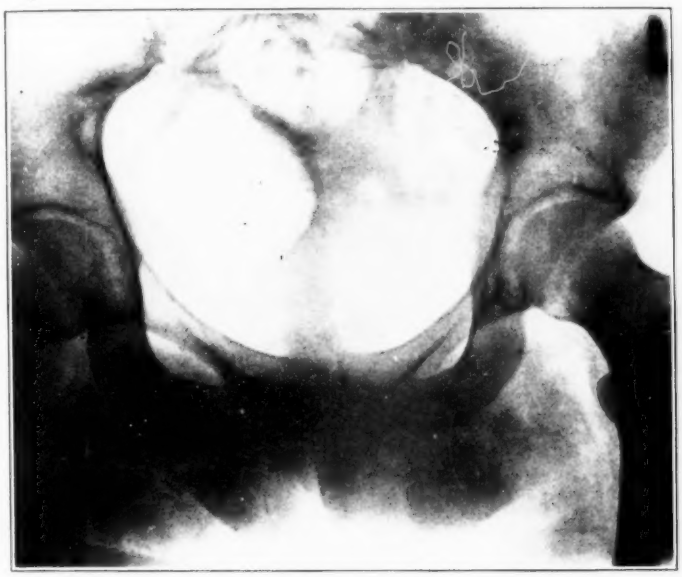

Fig. 8.—Case 2. After passage of the three calculi.

*Discussion.*—A review of impacted calculi in the ureter by A. H. Peacock,[8] covering 60 cases not associated with pregnancy, shows that the average time of an impacted stone is nine years, and that 67 per cent of all calculi are in the lower third of the ureter. Staphylococci were present in 47.5 per cent. McCarthy, Killian, and Chase[9] report proof of reflex anuria involving the otherwise healthy ureter, usually associated with calculus on the opposite side. Jeanbran[10] reports the successful recovery of a pregnant woman operated upon for calculus.

The third type of urinary reduction is represented by two very recent cases of eclampsia in the maternity ward of the University Hospital in young primiparae, each associated with recent hydronephrosis. For the past year we have been interested in the study of the kidney of pregnancy from the standpoint of differences in the two kidneys as determined by the cystoscope. We have found that, in addition to

the common mild degree of right side ureteral dilatation, there is at times a difference in the color, specific gravity, and cellular content of the urine of the two kidneys of toxic patients without any evidence of infection. The ureteral disease in the two following cases (Nos. 3 and 4), and the very frequent finding at autopsy of hydronephrosis and hydroureter in eclamptic cases, appear to be justification for our special investigation, which will be reported in the near future, to discover whether hydronephrosis of pregnancy is a cause of certain cases of acute toxemia and eclampsia.

CASE 3.—Mrs. G. S., aged sixteen years, colored, had been married one year. Her menses were normal. Her mother was subject to attacks of petit mal. The patient was admitted to the University Maternity 1/1/26 at 10:30 P.M., with the history of having delivered a full-term child three hours previously after an easy labor. Previous visits to the prenatal clinic at Southeastern Dispensary had disclosed no complications. Her condition was good until about twenty minutes after her delivery, when, during the expression of the placenta, she suddenly complained of violent frontal headache and a moment later had a generalized convulsion, following which she was extremely restless, confused, and had about four more convulsions. On admission she was extremely restless, unmanageable and confused. She was a normally developed, muscular girl. There was no apparent edema. Respirations were somewhat stertorous and accompanied by loud tracheal rhonchi. Blood pressure was 190/120, pulse 116, and temperature 99° by axilla. Her skin was moist. The bladder contained about 4 ounces of urine which boiled solid. During the first hour following her admission she had two convulsions. In the meantime 500 c.c. of blood were withdrawn by phlebotomy and 20 c.c. of 10 per cent magnesium sulphate were introduced into a vein. In addition she was given ½ grain of morphine subcutaneously. The stomach was washed out and three ounces of castor oil were introduced through a stomach tube. Following this she was comparatively quiet. Her blood pressure was 159/80. She was clearly oriented. In view of this and because her skin was actively eliminating, no further treatment by sweating followed at this time. She remained quiet for the next seven hours during which time she had only two slight convulsions.

At the end of this period, however, she suddenly had convulsions with increasing frequency and severity until 11 A.M. of the next day and despite active treatment during this period her convulsions remained uncontrolled. She remained unconscious and finally died during a convulsive seizure. Her treatment during this period consisted of a repetition of the phlebotomy, magnesium sulphate and in addition paraldehyde and normal saline solution. The blood urea nitrogen was 19, uric acid, 11.2.

Postmortem examination 1/2/26. Each kidney was apparently of normal size and position. The right ureter was dilated below the pelvic brim without stricture or stone; the left ureter was normal. Split kidney, left normal, right hydronephrotic; doubtful suggestion of degeneration.

Liver was of normal size; few subcapsular minute hemorrhages. Absence of normal "nutmeg" appearance and apparent early degeneration.

*Histologic studies:* There was not sufficient time for all organs to be sectioned. The right kidney showed severe tubular nephritis.

CASE 4.—Mrs. M. M., aged seventeen, colored, married one year, had a normal menstrual history; her last period was not known. She was admitted to the University Maternity 12/23/25 at 11:50 A.M., in a comatose condition. Pregnancy at about the ninth month. Convulsions had set in suddenly about five hours previously, recurring at frequent intervals until admission, when she was in complete

coma. Breathing was stertorous. Her skin was dry and hot. Temperature, 98.3°; pulse, 108; respiration, 32. There was very moderate edema of the ankles and pretibial areas. Blood pressure 175/95. Typical generalized eclamptic convulsions occurred at about fifteen minute intervals. Pregnancy advanced to about the ninth

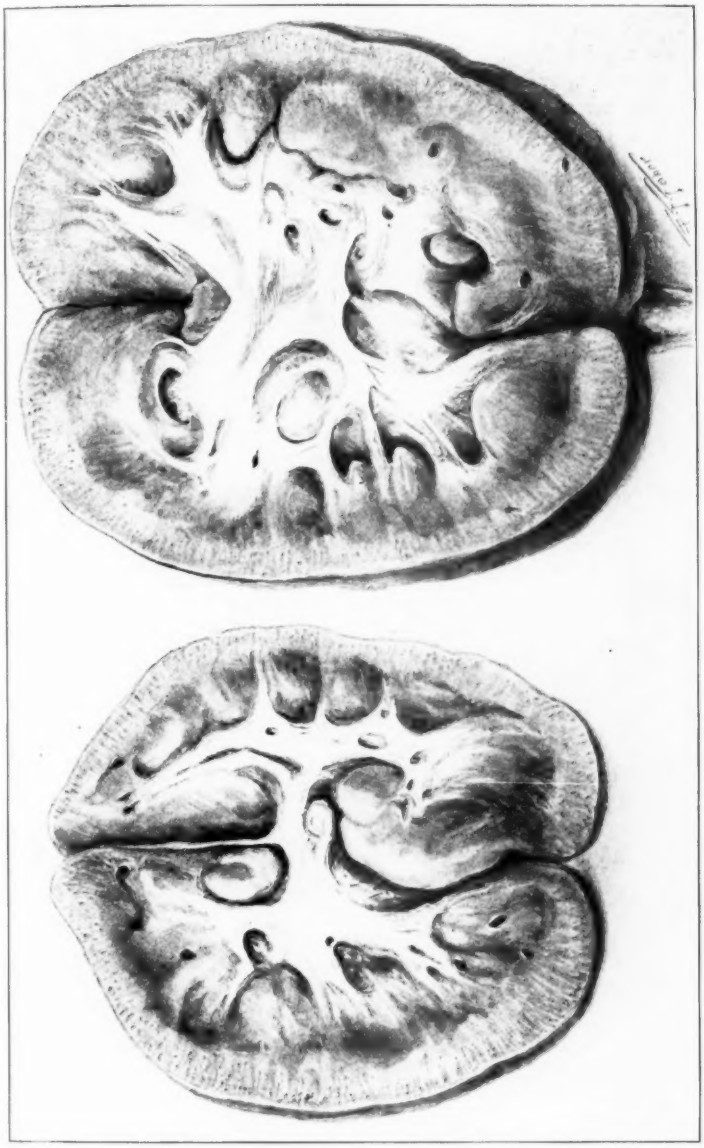

Fig. 9.—Case 3. Eclampsia, associated with hydronephrosis. (Dilatation of right kidney pelvis and ureter.)

month, head down, fetal heart not located, uterus tense, cervix closed and uneffaced, pelvis normal. General physical examination was otherwise negative. Twelve ounces of catheterized urine obtained; specific gravity of 1.006. It showed a faint trace of albumin and no casts. Blood urea nitrogen, 11 mg. per 100 c.c.

Patient was put upon an active regimen of elimination and sedatives, including gastric and colonic lavage, venesection, morphia, and 50 c.c. of 10 per cent magnesium sulphate in vein. By an oversight the patient was allowed to remain in one of her vapor baths for almost three-quarters of an hour, the nurse in attendance having carried out orders to the letter in allowing her to remain until the skin showed perspiration. The skin was found to be inactive, but the pulse had meanwhile risen to 156, and the blood pressure to 185/110. In spite of this rather active program, the patient continued to have convulsions, a total of twenty being registered in the first six hours after her admission.

Following a cesarean section the patient had a few more convulsions, but none after the first six hours. The mental state began to clear immediately. The blood pressure dropped to 130, at which point it remained for the next forty-six hours. The pulse remained at a high level, however, seldom dropping below 120. The urinary output in the first twenty-four hours was only 180 c.c. despite a good intake, and the peripheral edema did not appear to be taken up by her circulation. In the second twenty-four hours the urinary output increased to 900 c.c., but her pulse remained persistently high, and at the end of forty-six hours began to fail. She died forty-nine hours following operation.

After operation the blood count showed 5,900 white cells, hemoglobin, 60. The specific gravity of the urine was 1.012; it contained a heavy trace of albumin and hyaline and light granular casts. Blood urea nitrogen, 19; uric acid, 7.6.

*Autopsy report:* Double hydronephrosis (moderate) and hydroureter. Each kidney was extremely pale, showing slight yellowish tinge at cortex.

A very inaccurate table of average individual urinary output of the last fifty normal deliveries in the University Maternity without measuring intake, is as follows:

| | |
|---|---|
| First day | 29.7 oz. |
| Second day | 37.4 oz. |
| Third day | 34.7 oz. |
| Fourth day | 35.3 oz. |
| Fifth day | 36.2 oz. |

DeLee states that the amount of urine for the first eight days is 300 to 400 c.c. more than in the nonpregnant woman. Two of the above fifty cases passed less than 20 ounces of urine in the first day; therefore, it is important to instruct nurses of the necessity of keeping accurate record of urinary output at least until lactation begins.

On the pathology of hydronephrosis, H. P. Winsbury White[11] gives the following causes, from a study of 159 cases: Congenital, often associated with spina bifida and imperforate anus, and including horseshoe and ectopic kidney; inflammations; stricture; stone; pregnancy; abnormal renal vessels. In addition we would like to add that due to uterine fibroid, as seen in a case at the Philadelphia Hospital. Also, in addition to the urinary reduction from incarcerated retrodisplaced pregnant uterus and impacted fetal head, we wish to mention ureteral obstruction plus hematuria resulting from lateral saeculation of a pregnant uterus due to dense pelvic adhesions following myomectomy.

In conclusion, we wish to call particular attention to chronic pelvic passive congestion in pregnancy resulting in ureteral edema, to point out the necessity of proper regulations for the prevention of this difficulty, and to show the usefulness of the cystoscope in the diagnosis and treatment.

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1918 PINE STREET. (*For discussion see page* 768.)

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## INFARCTS OF THE PLACENTA: A STUDY OF SEVEN HUNDRED CONSECUTIVE PLACENTAS

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THE frequent occurrence of areas of so-called infarction has been repeatedly recognized by those who have examined a large number of placentas. Yet, a study of the literature must impress one with the fact that, even in recent times, there has been a wide divergence of opinion in regard to the nature, incidence, and significance of these masses. With this in mind we undertook certain studies of a series of placentas in an effort to throw more light upon the following points regarding infarcts:

1. Structure and materials entering into their formation.
2. Morphologic types.
3. Nature and etiology.
4. Incidence in normal and abnormal pregnancy.
5. Clinical significance.

The material and method of examination were as follows: Seven hundred placentas from consecutive deliveries, the majority at term and none before the eighth lunar month of pregnancy, were inspected, weighed, and measured. They were then hardened in 10 per cent formalin solution (4 per cent formaldehyde) for three to eight weeks, and cut in slices five to eight millimeters thick. Objects five millimeters or more in diameter, and suggestive of infarcts, were described

and later examined microscopically. Certain special studies were also made.

Four, more or less distinct, anatomic types of lesions were found deep in the placental tissue. Infarcts appearing on the fetal surface, including the marginata and margo varieties, were noted when at

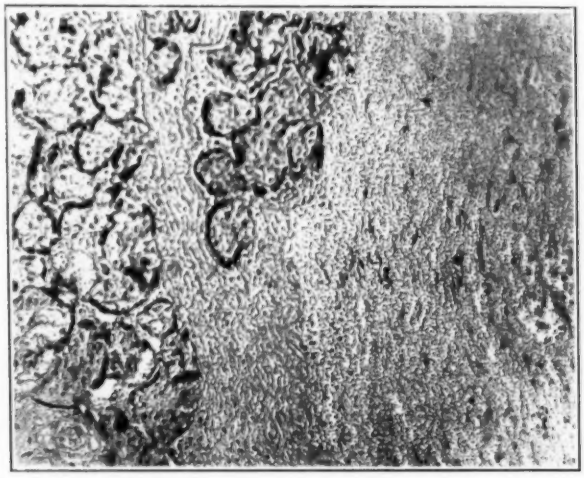

Fig. 1.—Infarct of our second kind (on right) laid down against an old infarct of the fourth kind (on left).

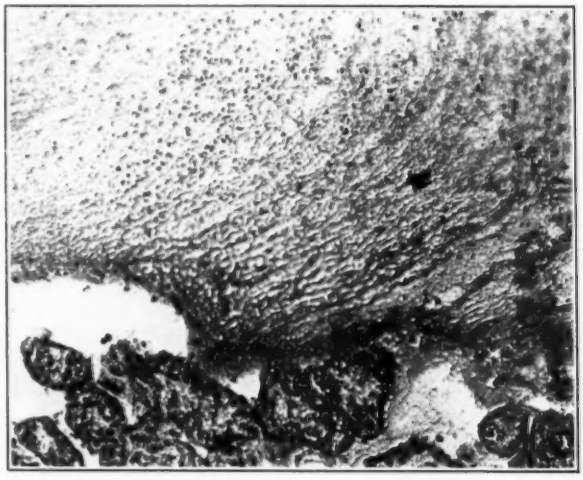

Fig. 2.—Part of the free margin of the same infarct of the second type shown in Fig. 1, showing very little involvement of villi. Note the laminated, thrombus-like appearance of the infarct.

least five millimeters thick. These structures usually belonged in our third group, although a few were of the first and second types, and were situated against the under surface of the thin chorionic membrane. Infarcts showing on the maternal surface were of all four kinds.

The nomenclature of these areas is in considerable confusion since many writers have objected, with some reason, to the old name of infarct and have suggested others as more suitable. We shall devote some discussion to this point under the different headings, but it may be stated here that we do not believe that an appropriate name

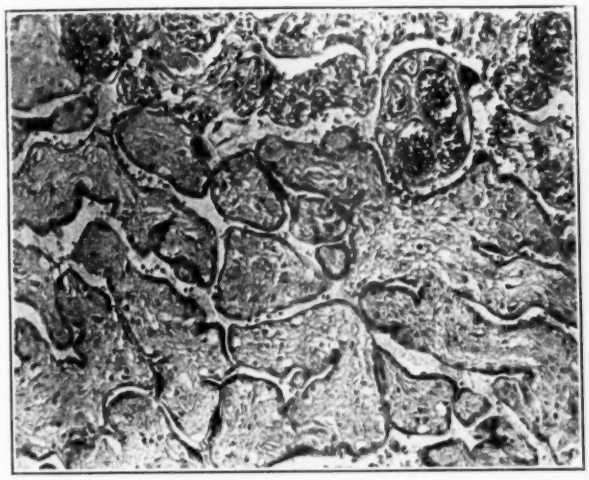

Fig. 3.—From the margin of an infarct of the fourth type, showing degenerated villi above and normal villi below. There is less fibrin than usual between the villi of this infarct.

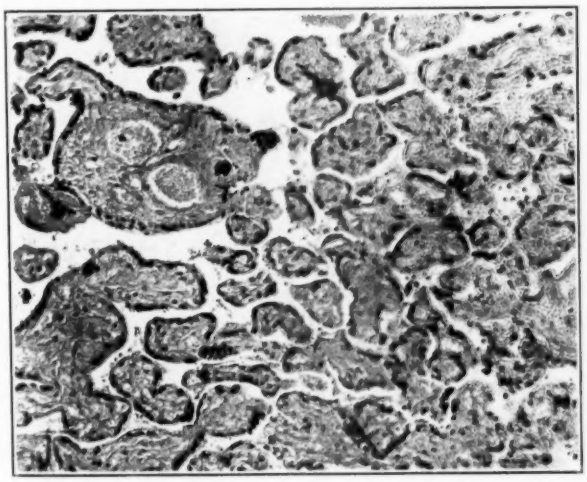

Fig. 4.—Placenta of a macerated baby. The degeneration of the villi resembles that found in infarcts of our fourth type.

for the whole group has yet been suggested and are inclined for the present to use the old and well-established designation, faulty as it is. In regard to the different kinds or varieties of infarcts, the terminology is in such confusion that in lieu of satisfactory names we shall refer to the four types of lesions by number as follows:

1. Infarcts of the first type are poorly defined, or even very irregular, pearl-gray formations occurring usually in the depths of the placenta but at times also near the surfaces and margins. They may vary from a few millimeters to several centimeters in width and occasionally are so large as to extend from surface to surface. We have never seen massive involvement as sometimes occurs in the last type. There are no striations, but there is often a mottled appearance near the outside due to the partial inclusion of small areas of normal placental tissue.

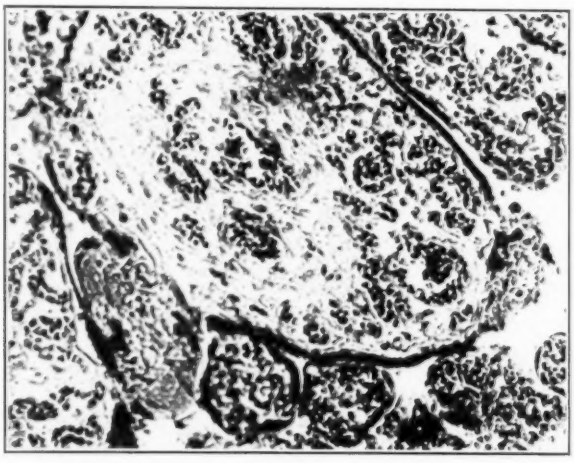

Fig. 5.—A villus showing several breaks in the continuity of the syncytium, with deposits of fibrin. The villous vessels appear normal.

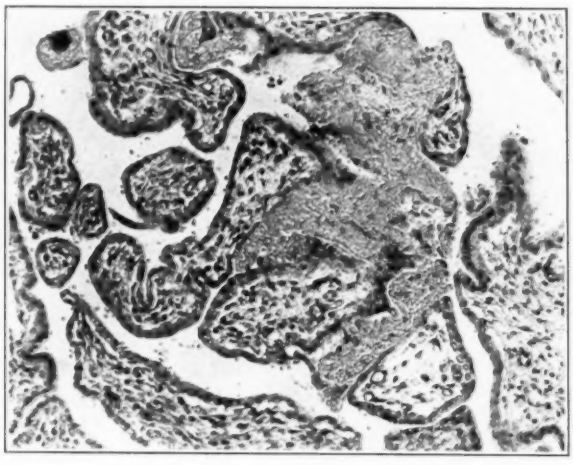

Fig. 6.—Placenta from abortion at the fourth lunar month, showing an early infarct of the first type.

Microscopic examination of an advanced infarct of this kind shows that at the edges there are broad projections of fibrin extending outward to outlying and nearly normal villi, while toward the center the structure becomes solid and consists more and more of degenerated shadows of villi surrounded by old fibrin, fragments of nuclei alone suggesting the original cellular structure. Evidently, the deeper portions are distinctly older, and examples are easily found showing development by

peripheral involvement of villi with fibrin projections from a nucleus of several villi matted together by fibrin.

2. Sharply demarcated, usually rounded or oval bodies, occasionally roughly quadrilateral, varying in diameter from a few millimeters to several centimeters and in color from red, brown, or almost black to pink or brick colored form the second class. The lighter colored masses are striated, often being made up of parallel dark and lighter striae. Infarcts of the second class are frequently surrounded by a distinctly lighter pseudocapsule and may often be found against, or partly surrounded by, infarcts of types 1 or 4. They may be seen singly, or in great numbers throughout the placenta, but usually deep in the placental tissue and seldom near the margins. When large and numerous, the condition has been called *placenta truffé*.

Microscopically, these infarcts are found to be composed of lamellae of fibrin and coagulated blood. Their color depends upon the number and state of preservation of the blood cells, the darker bodies and the dark streaks in the lighter showing densely packed and apparently unchanged red corpuscles. Sections taken at dif-

Fig. 7.—An infarct of the fourth type, showing a large branching villus which appears to be the stem for the villi involved in the infarct.

ferent levels show such an infarct always to be against, and in some instances partially surrounded by, a definitely older degenerative formation, usually an infarct of the fourth type, parallel to which the lamellae are laid down (Fig. 1). The rest of the circumference, as well as the deeper portion, shows practically no involvement of villi (Fig. 2). The fibrin on the free part of the periphery is frequently nearly devoid of blood cells, thus explaining the nature of the lighter colored pseudocapsule. In general, these infarcts bear a striking resemblance to fresh intravascular thrombi.

3. In the third group are striated bodies which so resemble those of the second kind in every respect, except color, that perhaps they should be included in that group. They are glistening or dull white, pinkish or definitely pink or brick colored as a whole or in streaks. In fact, infarcts of intermediate shades, with typical examples of this and the second group, are often found in the same placenta.

Microscopically they are seen to be composed essentially of lamellae of fibrin. In infarcts of a pink hue there are a few fragmented or degenerating and pale red blood cells between the lamellae. In accord with the darker gross appearance of

other of these bodies, the red cells may be present in such numbers that the infarcts are obviously intermediate forms between the second and third types.

4. Round, oval, or pyramidal bodies usually found near the margin, less often deep in the placental tissue, sometimes near the maternal surface, seldom near the fetal surface constitute the last group. They vary in size from nodules a few millimeters in diameter to masses involving one or several cotyledons. We have once seen approximately one-fourth of a placenta thus infarcted, and on another occasion the entire substance of a succenturiate lobe. Older infarcts of this kind are usually a uniform dull white or light yellow, and the edges, though often appearing slightly uneven or fuzzy, are sharply contrasted with the surrounding placental tissue. The younger forms, as occasionally seen, are pink or even dark reddish brown and are usually mottled. Frequently, these infarcts are found against those of the second and third types (Fig. 1).

Microscopically, these bodies are seen to be composed of closely packed villi with, usually, a thin layer of fibrin between (Figs. 1, 3, and 7). It is characteristic of these infarcts that all the villi in each have reached about the same degree of degeneration. In the more recent forms there is likely to be very little or no fibrin, and the villous vessels are filled, or even widely distended, with apparently normal blood, while the syncytial epithelium appears normal. However, the villous stroma cells stain lightly and are poorly defined, while the nuclei are pyknotic and shrunken. The periphery, at this stage, is somewhat irregular but quite definite, due to the sharply different appearance of the normal and infarcted villi, there being no intermediate forms (Fig. 3). In the advanced stages the blood cells in the villous vessels are faded and disintegrated, while the stroma has lost all evidence of its cellular structure except for a few fragmented nuclei. The syncytial epithelial covering persists longer, at first seeming even to proliferate, but eventually it too degenerates, and finally, there is the picture of structureless but still distinctly outlined villi. The periphery in the old forms is often marked off by a narrow zone of infarction of the first type, or it may be against a body of the second or third kind.

Besides these so-called infarcts, certain other bodies were found. Cysts of the chorionic trophoblast were common, as were also small masses of degenerated trophoblastic cells. The latter occasionally seemed to have served as the starting point for infarcts of the first, second, and third types, but otherwise were of no importance. Occasional angiofibromas were found, but they had no relation to infarct formation. Of frequent occurrence were small, irregular areas of dark blood, some gelatinous in consistency, which upon microscopic examination resembled structureless blood clots, with normal villi irregularly surrounding and dipping into them. In many instances the blood cells had settled toward one side. Although McNalley and Dieckmann speak of these as a type of infarct, we believe them to be only blood which has coagulated between the villi after delivery. In sections taken from fresh placentas they are rarely present. Such is also the case with certain small, empty, round or oval cavities, without capsule and bounded by normal villi, which are probably due to bubbles of air, or perhaps gases from bacterial activity before fixation was complete.

## NATURE AND ETIOLOGY OF INFARCTS OF THE PLACENTA

In this study we have confined direct reference to the literature principally to that of the last twenty-six years, since J. Whitridge Williams thoroughly covered the ground up to 1900. We have also consulted more recent reviews of the older literature by Giese, Kalima, Clemenz, Strachan, and others. It now seems established that placental infarcts are composed essentially of chorionic villi and maternal

blood. It is, however, to be noted that in 1908 Brindeau and Nattan-Larrier expressed the belief that in eclampsia there could be found infarcts formed from fetal blood which had escaped into the intervillous spaces by rupture of the villous capillaries. Some authors admit that a small amount of degenerated decidua may sometimes be included in infarcts, especially in those at the margin, but none agree any longer with Steffeck that degenerated decidua forms the basis of placental infarction. Work along embryologic and histologic lines has given fairly conclusive evidence that "decidual islands" and "septa," as well as the top layers of the retroplacental decidua, are really composed of fetal trophoblast or its degeneration products, though opinion to the contrary is still held by McNalley. In any event, it may be safely assumed that the principal constituents of placental infarcts are degenerated villi and maternal blood elements.

The varying proportions and state of preservation of the materials in infarcts account in large part for the differences in gross appearance. Histologically there are other characteristics which probably have some significance in regard to the formation and development. Consequently, the types will be considered separately, though it is true that some of the masses are apparently made up of a combination of the various kinds of infarcts.

*Type 1.*—Infarcts of this kind have received considerable attention, and the concensus of recent opinion is that they are the product of the coagulation of maternal blood elements (mainly fibrin) with the involvement of adjacent villi. Schickelé has suggested the name "fibrin nodule" and Clemenz "white necrosis" as more appropriate. Certainly, strictly speaking, these bodies are not infarcts. Although the idea is not entirely of recent origin, Hitschmann and Lindenthal, Schickelé, Kalima, Clemenz, and others have emphasized that their formation depends upon the same conditions as does intravascular thrombosis, namely, exposure of tissue by loss or degeneration of its anticoagulative covering, and a slow blood stream. The placental space with the villi dipping down is looked upon as a blood vessel, the trophoblastic syncytium serving the same function here as endothelium in blood vessels.

The relative importance of the two factors is still a matter of debate. Indeed, there are those who contend that one, or the other, alone is responsible for the initiation of the lesions. From the evidence at hand, we are inclined to believe that either condition may be the cause of these infarcts, but that in most instances both factors play a part, or, at least, are present in the placenta near term.

Huguenin is one of the many authors who emphasize the importance of areas favoring coagulation. He lists the tissues which, when exposed, would cause deposits from the blood as chorionic connective tissue, cells of the Langhans layer, fibrin, decidual cells, and already

deposited blood materials. Obviously, the last material may only be present secondarily. Hitschmann and Lindenthal, and others before them, deny that the decidua has any place in infarct formation. Perhaps the older references to decidual cells were to what we now believe to be the fetal cells in the so-called "decidual islands" and top layers of the decidua. Chorionic connective tissue and Langhans' cells may sometimes be exposed by the loss of the syncytial covering, but usually in such event these tissues have already undergone more or less degeneration with the formation of a layer of fibrin-like material. Langhans, and later Biland, described this fibrin layer beneath the chorionic membrane and attributed its formation to degeneration of Langhans' epithelium cells. It is also found on the decidual surface and in the position formerly occupied by the Langhans' epithelium of the villi. It is ordinarily considered, when exposed by the loss of the syncytial covering, to be the basis for deposits of maternal blood fibrin. Langhans noted an association of the fibrin layer formation with syncytial degeneration, and since his time considerable work has been done to determine which process takes place first, and also whether there is a causal relationship.

Ackermann, Eden, and Williams (1900) believed obliterative endarteritis in the villi, and frequently periarteritis, resulting in coagulation necrosis of the portions of the villi just beneath the syncytium, to be the first step. The syncytium, though probably partially dependent on the maternal blood for nourishment, was thought to degenerate subsequently, perhaps as a result of suspension of its function of exchange (Williams, 1900). The vessel changes were found by Williams (1900) to be most marked in the medium-sized villi. Eden believed them to be an evidence of senility of the organ. He found them more frequently at the periphery, comparatively few of the branches being involved. He furthermore stated that he could differentiate such changes from the ordinary postpartum contraction of villous arteries. Certain extreme instances of obliterative endarteritis of villous vessels, but without much apparent relationship to infarction, have been reported (Von Franqué, Rielander, Müller). Fraser, moreover, believes that his work on injection of placental vessels confirms the hypothesis of Ackermann, Eden, and Williams.

This view has been vigorously challenged. In fact, Hitschmann and Lindenthal, Clemenz, Giese, and others denied that they had ever seen such arterial changes. Schickelé stated that to him the picture of arteritis in the villi was not clear-cut, and that certainly there was no such association with necrosis in the placenta. Huguenin pointed out the similarity of the villous arteries to those of the cord and believed the appearance of both to be due to postpartum cessation of circulation.

Hitschmann and Lindenthal, Clemenz, Young, Strachan, McNalley,

and others contend further that the villous epithelium and at least part of the stroma are dependent on the maternal blood for nourishment; and that, therefore, degeneration of the villous epithelium would not result from disturbance of blood supply in the villous vessels, even should this occur. Evidence upon which their opinion is based may be summarized as follows:

1. In young ova before fetal vessels are formed the villi grow rapidly, apparently being nourished directly by the maternal blood.

2. In hydatid mole the villi grow rapidly after death of the fetus and there is no trace of fetal vessels. The same is true of chorioepithelioma.

3. Young reports an instance of tubal pregnancy showing degeneration of the villous trunks but with good preservation of villous tips near the tube wall where the maternal circulation was unimpaired.

4. In syphilitic placentas, with the villous vessels few in number or even entirely absent, there is little more infarction than in the normal organ.

5. The placental villi develop over the decidua basalis; while elsewhere the villi degenerate as the growth of the ovum thins out the reflexa and thus reduces its maternal blood supply.

6. Villi may be deported through the maternal veins and remain in distant organs for some time without change in their structure.

When this evidence is examined, it is found far from convincing. In the first place, in early pregnancy not only the villi but also the embryo grow rapidly, although the latter is not bathed by maternal blood. Obviously, conditions are not the same as during the latter part of pregnancy. The fact that the placenta forms over the basalis does not necessarily mean that villi are nourished directly by maternal blood but rather that they reach their full functional development at the point where most nourishment may be obtained. Hydatid mole and chorioepithelioma are now known to arise as abnormal growths of the chorionic epithelium. In the mole there is degeneration of the stroma. Both give a tumor picture in no way resembling normal villi. Hitschmann and Lindenthal presented the evidence in regard to the frequent normal appearance of deported villi, but in another part of their paper they refer to the opinion of Weigert and Cohnheim that cell death is not immediately evident microscopically, and that the changes usually ascribed to death really take place subsequently. And Young states that dead villi do not become evident as infarcts for ten or eleven days. In regard to the third argument, we cannot express a definite opinion for early pregnancy, but in placentas from late pregnancy the villi soon show evidences of degeneration, often within a few days after fetal death. This is true even when the intervillous spaces are open, and it may be taken for granted that circulation of maternal blood has continued (Fig. 4). We can present no objection to the evidence under the fifth division except to cite the opinion of Huguenin that syphilis does lead to increased infarction, though he thinks the cause is not the arteritis but rather

the difficulty in exchange, due to the increased stroma, which finally leads to functional atrophy of the syncytium. None of the placentas in our series showed syphilitic changes.

On the other hand, there are certain reasons for believing that chorionic tissue may be nourished through the fetal circulation. Müller described three placentas showing extreme dilatation of the capillaries and beginning degeneration of the villous branches in association with arteritis and thrombosis of larger villous vessels. In one case the thrombosis was the result of inflammation, but in the others the cause was not clear. Of greater importance, we believe, are the reports of certain placental tumors. Goodhart and Calderini described two large angiofibromas of the chorion which were connected to the placenta by vascular pedicles but were not attached to the decidua. Margeson found a tumor mass 10 by 6 by 3 centimeters lying in the membranes, and thus separated from the uterus, which as its only attachment had a six centimeter vascular pedicle to the placenta. We have seen two chorioangiofibromas consisting of lobes so tightly pressed together that no maternal blood could have flowed between, and yet, syncytial investment of the lobes, though nourished as it must have been through the fetal circulation, was perfectly preserved. Certainly, this is indisputable evidence that, under some circumstances, at least, tissues of chorionic origin can be nourished by the fetal circulation alone.

Recently, many authors have considered the thinning, degeneration, and final loss of the syncytium (and also the consequent fibrin deposition) as primarily physiologic and probably as an evidence of senility of the placenta (Clemenz, Hitschmann and Lindenthal, and others). Huguenin thinks that expansive growth of the villi causes thinning of the syncytium and breaks in its continuity, which act as the initial points for the deposition of fibrin. Certainly, as pointed out by Kalima, many small villi can be found with breaks in the syncytium and secondary fibrin deposits, but with apparently normal vessels (Fig. 5). No explanation has been offered for this same finding, which is frequent, in early placentas (Fig. 6).

As stated before, the other usually accepted factor in infarct formation is the slowness of the maternal blood stream between the villi. Although Huguenin, Biland, and Kalima contend that deposition of fibrin cannot take place without a break in the continuity of the syncytium, Clemenz and others, though believing that both factors are usually active, point to certain instances of intervillous thrombosis without any evidence of damage to the syncytium of the involved villi. Giese held the extreme view that stasis in the maternal blood stream was always the primary factor, and that degeneration of the syncytium was consequent upon this. Young has been supported by McNalley in his opinion that infarcts are due to a local disturbance in the circulation of maternal blood. This presupposes that there is not a general circulation of

maternal blood through the placenta, but that groups of villi are dependent on more or less isolated systems, demarcated probably by "decidual septa." This is in accord with Bumm's hypothesis, which, however, has been convincingly contested by Kermauner and Wieloch. We found no septa of any size at the placental site of a uterus with placenta in situ, which had been removed at the sixth lunar month. Many authors now consider the intervillous placental space to be a sort of lake of maternal blood with a slow current as a result of its greater volume in comparison with that of entering vessels. Toward term this stasis may become more marked by reason of plugging of uterine veins by giant cells (Eden) or deported villi (Giese), or to obliterative changes and thrombosis in the arteries and veins, especially in the presence of toxemia of pregnancy (Rohr from Hitschmann and Lindenthal, Williams [1917], Schwarz and McNalley).

Under special conditions, it has been suggested that there are changes in the blood itself which favor infarct formation. For example, Brindeau and Nattan-Larrier point out that Bar found increased coagulability of the maternal blood in toxemia. Kworostansky believed such a condition to be responsible for increased placental infarction in heart failure as well as in nephritis.

*Type 2.*—As stated under their description, these bodies closely resemble rapidly formed, intravascular thrombi, their structure consisting of layers or lamellae of fibrin in which red and white blood cells, but rarely any villi, are enmeshed. Serial sections show the lamellae at one side to be laid down against definitely older infarcts of the first or fourth types, or degenerated areas beneath the chorion or above the decidua. The infarcts sometimes partly cover or cap the thrombus masses (Fig. 1), but the latter have most of the periphery free (Fig. 2). This part of the periphery often presents a layer of more compact fibrin with few blood cells, thus giving the gross picture of a lighter colored capsule. The presence in these nodules of blood from the fetal vessels (Brindeau and Nattan-Larrier) has not been confirmed. Probably they are formed by rapid coagulation of blood in layers against older degenerated areas, normal villi being pushed back as the masses increase in size. Von Franqué referred to reports by Prochownick, Bumm, and others and also reported two such cases himself as "thrombosis sinum placentae." Huguenin noted their resemblance to thrombi of the heart and great vessels. Haffner objects to the name "infarct" as inappropriate. Nor, should the names hematoma, hemorrhage, or apoplexy (Brindeau and Nattan-Larrier) be used, since these masses are formations in the intervillous placental space, which functions as a blood vessel (Schilling from Von Franqué, Williams, 1923). They should not be confused with retroplacental blood extravasations resulting from premature separation of the placenta or uteroplacental apoplexy, as in our experience the latter are without structure and resemble ordinary blood clots.

*Type 3.*—These bodies are identical in structure to those of the second group, except for a much less number, or even an entire absence, of blood cells between the fibrinous lamellae. Haffner considered them in the same class with the darker structures, ascribing the difference in color to a varying content of red blood cells. Brindeau and Nattan-Larrier noted that the older masses were lighter in color. Several authors have pointed out that newly infarcted areas corresponding to our first type included varying numbers of blood cells which later faded and disappeared (Kalima). McNalley and Dieckmann think that, similarly, degeneration of blood cells in the dark striated bodies results in the lighter ones. We have confirmed their findings in regard to the frequent existence of what appear to be intermediate forms between the dark and the white masses. Sometimes this transition may be apparent in the different parts of the same thrombus.

*Type 4.*—Ordinarily, these infarcts, especially the older ones, have not been differentiated from those of the first group. However, Eden noted their distinctive structure and speculated as to their origin. He spoke of them as nonfibrinous infarcts. Williams described these bodies, and Brindeau and Nattan-Larrier saw them against infarcts similar to our second kind. Young called the early ones "red infarcts" but noted that they faded later, the old ones becoming white.

Eden supposed the development of such an infarct to be due to a local failure of the maternal circulation supplying the area of placenta occupied by the infarct. He assumed that, as the pressure of the blood was lowered, it no longer held the villi apart but permitted them to collapse against each other. Young supported this hypothesis and in some cases attributed the disturbance in circulation to retroplacental and intraplacental hemorrhage. However, in at least some of his colored illustrations the infarcts have existed for a sufficient time to become light brown or nearly white, while the hemorrhages are still dark and thus probably are more recent formations. In order to accept Eden's explanation we must assume that the maternal blood circulation in the intervillous spaces is carried on by a number of independent systems, an idea against which Kermauner and Wieloch have presented convincing evidence, as stated above. We have demonstrated by serial sections that younger infarcts of this type may be found deep in the placental tissue and entirely surrounded by apparently unaffected villi. Fig. 3 shows part of the periphery of such an infarct, as does Young's Fig. 4 of Plate II. It is difficult to conceive of a unit of intervillous circulation so circumscribed that its failure can cause necrosis of villi in a well-defined group and not affect those directly adjacent on all sides. Indeed, the fact, noted by others and confirmed by us, that the syncytial investment of the villi persists even after there is demonstrable necrosis of the stroma suggests that the primary disturbance of circulation was in the fetal vessels rather than in the intervillous space.

In connection with the cause of these infarcts it is to be noted that in the younger bodies the villi have undergone about the same degree of degeneration. In general this holds also for the older ones, except that at the periphery there is being formed infarction of the first type, but evidently secondarily. Apparently, then, these infarcts are not built up gradually, but all the villi are affected simultaneously. Also, we have noted several times a large branching villus entering in a manner to suggest that it was the villous stem for all villi in the infarct (Fig. 7). All the tissue that remained of three such infarcts was cut in serial sections without other large villi being found. Only a few sections from infarcts of the first type are required to prove that many villous stems and their terminal branches are involved. Next, as a check, all the tissue of two small infarcts of our fourth kind, one five millimeters in diameter and the other slightly smaller, were cut into serial microscopic sections. In each instance all the branches to which the degenerated terminal villi were attached could be traced to one entering villous stem. All the vessels were thrombosed; but because of the difficulty in identifying such small bodies suitable for serial sections, both infarcts selected were so degenerated that it was impossible to determine definitely whether thrombosis was primary in the smaller villi or in the stem. However, the histologic evidence strongly suggests that these bodies represent instances of true infarction due to a disturbance of the fetal-placental circulation.

## INCIDENCE AND CLINICAL SIGNIFICANCE

From early times the frequent occurrence of infarcts of the placenta has been noted, but with wide discrepancies in the figures of incidence. Williams (1900) attributed this to differences in methods of examinations and in criteria. He saw microscopic infarcts in all placentas at term and found bodies at least a centimeter in diameter in 63 per cent, including the placenta marginata type. More recently Haffner and Ravenstein, employing methods somewhat comparable to ours, reported an incidence of 42.6 per cent and 72 per cent from 400 and 260 placentas, respectively. Among our 700 consecutive placentas there were 474 or 67.7 per cent with infarcts of some kind measuring at least five millimeters in diameter.

Kalima found slightly more infarcts in placentas from multiparas than in those from primiparas, and attributed the difference to a greater likelihood of the development of stasis in the uterine vessels of multiparas. The percentage incidences in our 700 placentas (Table I) fail to show such a relationship, the differences being so slight as to make their significance questionable. Also, apparently, age has no influence on the development of infarcts.

The older authors considered the presence of albuminuria to be an important factor in infarct formation. Williams (1900) confirmed them

TABLE I

PERCENTAGE INCIDENCE OF PLACENTAL INFARCTS IN RELATION TO AGE AND PARITY OF PATIENTS

| NUMBER OF PLACENTAS | ALL PLACENTAS | YOUNG (UNDER 30 YR.) | OLD OVER 30 YR.) | PARA I | PARA II | PARA III | YOUNG PARA I | OLD PARA I | YOUNG PARA II | OLD PARA II | YOUNG PARA III | OLD PARA III+ |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | 700 | 402 | 298 | 364 | 214 | 122 | 263 | 191 | 105 | 109 | 34 | 88 |
| Type 1 | 13.1 | 13.9 | 12.1 | 12.1 | 15.9 | 11.5 | 12.5 | 10.9 | 17.1 | 14.7 | 14.7 | 10.2 |
| Type 2 | 25.7 | 26.9 | 24.5 | 24.5 | 26.6 | 28.7 | 25.5 | 21.8 | 28.6 | 24.8 | 32.4 | 27.3 |
| Type 3 | 33.9 | 32.1 | 36.2 | 32.1 | 33.2 | 40.2 | 29.7 | 38.6 | 32.4 | 33.9 | 50.0 | 36.4 |
| Type 4 | 19.1 | 19.4 | 18.8 | 20.6 | 17.3 | 18.0 | 20.2 | 21.8 | 17.1 | 17.4 | 20.6 | 17.0 |
| Infarct of some typ | 67.7 | 66.9 | 68.8 | 66.8 | 67.3 | 71.3 | 65.4 | 70.3 | 67.6 | 66.9 | 76.5 | 69.3 |

in regard to the relationship of red infarcts (our second type) to nephritic toxemia, but McNalley and Dieckmann found these bodies very frequently also in placentas from normal pregnancy. Kalima held that toxemia of pregnancy caused the formation of large infarcts. Young believed infarcts corresponding to our fourth type to be of importance in the etiology of albuminuria. Haffner, and later Ravenstein, denied that there was any connection between albuminuria and the incidence of infarcts. In our 700 placentas there were 45 from pregnancies complicated by so-called toxemias of pregnancy, as indicated by albuminuria associated with hypertension. Clinically there were 37 cases of preeclamptic toxemia, 5 of eclampsia, and 3 of nephritis. Not only the incidence of all types of infarcts (Table II) but also their size and

TABLE II

PERCENTAGE INCIDENCE OF INFARCTS IN PLACENTAS ASSOCIATED WITH TOXEMIA OF PREGNANCY

| | ALL PLACENTAS | PREECLAMPSIA | ECLAMPSIA | NEPHRITIS |
|---|---|---|---|---|
| Number Placentas | 45 | 37 | 5 | 3 |
| Type 1 | 17.8 | 18.9 | 0 | 33.3 |
| Type 2 | 31.1 | 29.7 | 40 | 33.3 |
| Type 3 | 37.8 | 40.5 | 20 | 33.3 |
| Type 4 | 42.2 | 40.5 | 60 | 33.3 |
| Infarct of some type | 86.7 | 83.8 | 100 | 100 |

TABLE III

PERCENTAGE INCIDENCE OF SMALL AND LARGE OR NUMEROUS INFARCTS

| | NORMAL PREGNANCY | PREGNANCY WITH TOXEMIA |
|---|---|---|
| Number Placentas | 655 | 45 |
| No infarcts | 32.8 | 13.3 |
| Small infarcts | 54.4 | 60.0 |
| Large or many infarcts | 11.75 | 26.7 |

number were greater (Table III). Because of the fact that infarcts are found in the majority of placentas from normal pregnancy, and for other obvious reasons, it seems probable that their higher incidence with albuminuria is a consequence rather than a cause, as believed by Young. The increased infarction with the toxemias may be due to intervillous stasis consequent upon the more marked changes in the uterine vessels (Kalima, Schwarz and McNalley).

It has long been held that extensive infarction of the placenta might lead to such a disturbance in circulation as to result in a poorly developed child, and even its premature delivery or death in utero. Fehling believed that toxemia of pregnancy led to extensive infarction which in turn caused the death of the child (Williams, 1900, Kalima). In our series the number of stillbirths (eighteen) does not permit a statistical study of much value in regard to their relationship to the occurrence and size of infarcts. However, it is noteworthy that among

the nine stillbirths, whose cause was debatable, only two were associated with more than a slight infarct formation. Five were associated with toxemia of pregnancy, but in only one instance was there marked infarct formation; hence the inference that marked infarcation bears no frequent or distinct relationship to stillbirths, either primarily or secondarily as a result of the presence of toxemia. Nor, if we accept our figures for normal pregnancy, do infarcts of the placenta, even when large, interfere with the growth of the fetus (Table IV). With toxemia,

TABLE IV

AVERAGE WEIGHT IN GRAMS OF BABIES IN RELATION TO INFARCTION OF THE PLACENTA

| | NORMAL PREGNANCY | TOXEMIA | TOXEMIA MINUS INDUCED LABOR |
|---|---|---|---|
| Number babies | 655 | 45 | 41 |
| No infarcts | 3305 | 2865 | 2865 |
| Small infarcts | 3375 | 2975 | 3030 |
| Large or many infarcts | 3315 | 2700 | 2880 |

on the other hand, the average weight, though reduced in all groups, showed a great reduction for the children associated with large or numerous placental infarcts. However, when babies born after induction of premature labor were eliminated from the calculations, the figures are without much significance (Table IV). Then, in these 700 consecutive deliveries there is little evidence that infarcts of the placenta, even when large, bear any relationship to the welfare of the fetus, either in normal pregnancy or when so-called toxemia of pregnancy exists.

## SUMMARY

So-called infarcts of the placenta are of four kinds, but are all composed largely of degenerated villi and elements from the maternal blood. In three types the formation resembles intravascular thrombosis and depends upon stasis of the maternal blood flow in the intervillous placental space, and the existence of areas denuded in some way of their anticoagulative syncytial epithelium. The fourth kind probably represents a simultaneous involvement of all the branches of a stem villus due to disturbance in the fetal-placental circulation.

In 700 carefully examined placentas, which were delivered consecutively, there were infarcts of some kind in 67.7 per cent, there being no relationship of occurrence to age or number of pregnancies. All types were more frequent in placentas associated with toxemia of pregnancy, as was also extensive infarction. The presence of infarcts had little or no influence on the welfare of the child.

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# THE CLINICAL SIGNIFICANCE OF THE SEDIMENTATION TEST AS A DIAGNOSTIC AND PROGNOSTIC SIGN*

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DURING the last decade there has been a steady trend toward greater conservatism in the treatment of pelvic infections for, with an increasing knowledge of the physiologic pathology which takes place in these lesions, whether of a gonorrheal, postabortal, or postpartal origin, we recognize more and more, that the patient who recovers does so by a resistance due to her natural forces, i.e., by the production of a leucocytosis, tissue cell proliferation, and the formation of antibodies which overcome the invading organisms in the tissue fluids. Furthermore, it is these body fluids that dispose of the toxins.

Surgical attack is being limited to the drainage of local pus foci and to the removal of the results of these infections when they have reached the quiescent stage. Even here there is a proper and an improper time for intervention.

It is in the hope of adding something to our clinical knowledge, that may help us to determine the presence of latent infections and when to operate and when not to interfere, that we have undertaken this study.

During 1925 and 1926, each patient admitted to the gynecologic wards of the Long Island College Hospital has had repeated sedimentation tests done, in addition to the routine laboratory and clinical data. Hence the significance of the sedimentation rate in almost every gynecologic condition has been checked, grouped, and correlated. The usual preoperative routine includes a detailed history, a complete physical examination, an examination of the urine, study of the blood chemistry, a Wassermann reaction, a hemoglobin estimation, a complete blood and differential count, kidney function tests, temperature, pulse and blood pressure readings.

Even with these data we have at times been unable to determine the presence of latent infections and to prognosticate the postoperative course of patients when operation was to be performed.

It is admitted that a rapid sedimentation time spells infection, but

*Read at the Fifty-first Annual Meeting of the American Gynecological Society, Stockbridge, Mass., May 20, 21, and 22, 1926.

it does not necessarily mean that this infection is in the pelvis. It is, however, a danger signal which should be heeded in considering the time for operation, even when temperature and blood count are favorable. Furthermore, it has been shown that the usefulness of the test depends on its *frequent repetition* and its correlation with the other known clinical reactions, as the blood, temperature, and pulse. Therefore, from this study we hope to show:

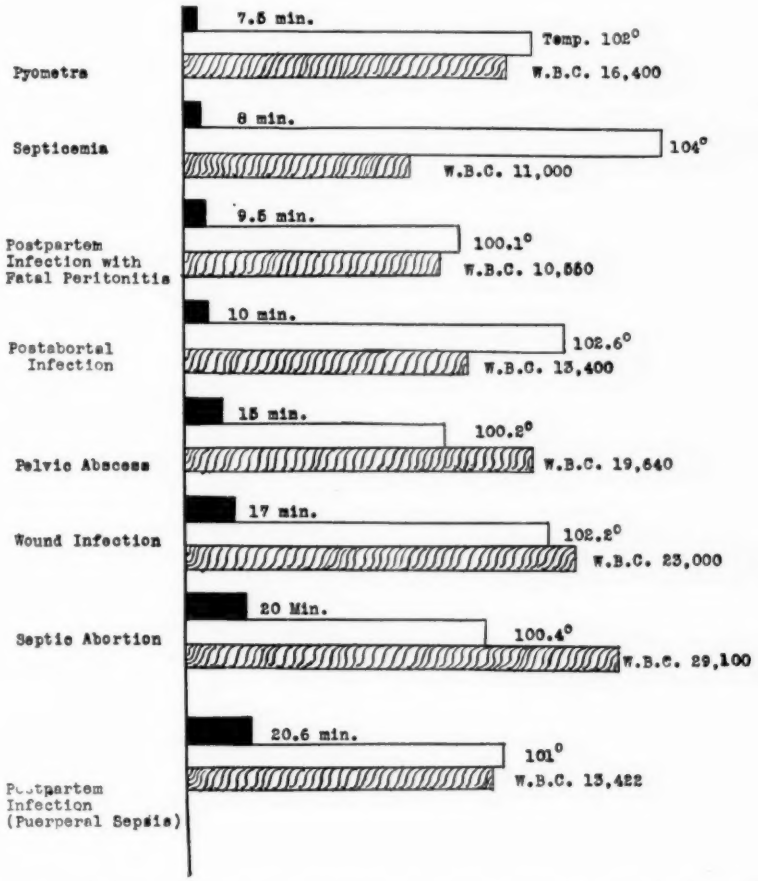


Chart I.—Table showing average sedimentation rates, leucocyte count, and temperature in groups of acute cases.

1. That the sedimentation test when repeated daily, or two or three times a week in any case of pelvic infection, and when considered in conjunction with the pulse rate, temperature curve, and the leucocyte and differential count, is a diagnostic as well as a prognostic sign of great importance.

2. That while a single test may show the presence of infection, it is not a fair index of the potential danger of said infection when the

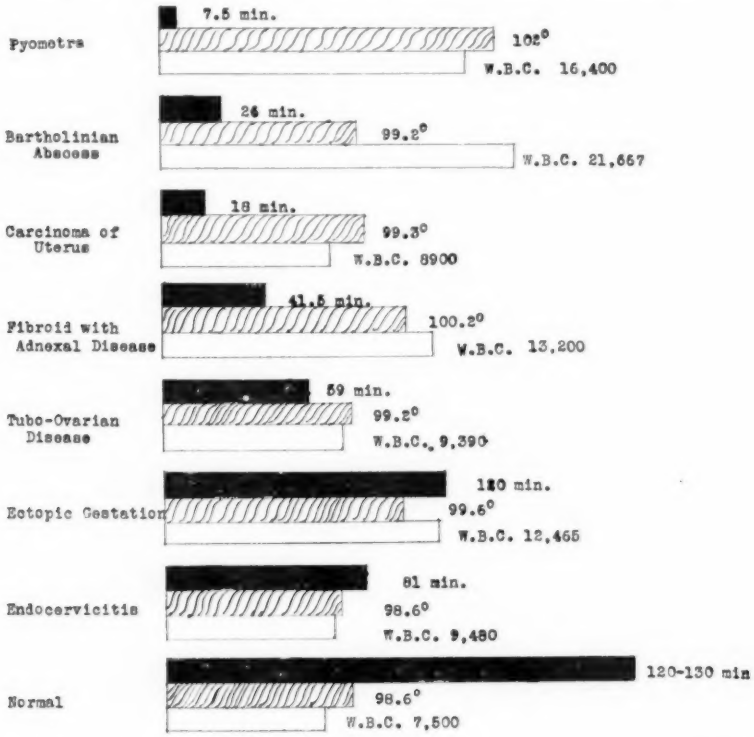


Chart II.—Relation of the average sedimentation time, temperature, and leucocyte count in several groups of cases. Compared with the average normal sedimentation time.

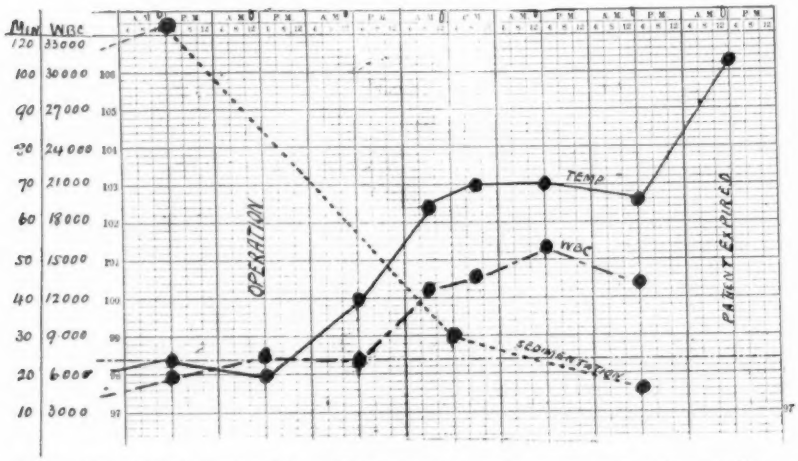


Chart III.—Fatal Peritonitis: This chart reveals a normal sedimentation rate, temperature, and white blood cell count prior to operation. Following operation the temperature began to rise first; the leucocyte began to rise slowly, and the sedimentation rate was very rapid. The sudden drop in rate, together with the increase in temperature, indicates a bad prognosis. In the follow-up of this case it will be noted the temperature became remittent in type varying from 100° to 103° and 106.2° premortem. The sedimentation rate became more rapid, while the leucocyte curve remained at about the same level. From this case, one would be inclined to believe that the sedimentation test was the more sensitive, but we feel that the combination of the three clinical methods gave more information than any one finding.

barriers are disturbed or broken down by manipulation or operative procedure, unless it is repeated and correlated with other clinical data.

*Technic:* All of the tests were done by one person so that there could be but little chance of technical error. The technic employed in this investigation was that of Linzenmeier as modified by Friedlaender. Hard glass tubes 5 mm. in diameter and 6.5 cm. in length with a capacity of more than 1 c.c. were used. The tubes were marked at the 1 c.c. level and at 6, 12, 18, and 24 mm. respectively below. Eight-tenths c.c. of blood was drawn directly from the vein into a Luer tuberculin syringe which contained 0.2 c.c. of a freshly prepared 5 per cent solution of sodium citrate. The blood and citrate solution were shaken until thoroughly mixed. The mixture was then placed in a sedimentation tube and allowed to stand at room temperature. The time was noted when the mixture was placed in the tubes, and observations were made from five minutes and upwards as found necessary. The time was noted when the line of demarkation between the erythrocytes and the plasma reached 6, 12, and 24 mm. respectively. The readings used in this report correspond to the time for the line of demarkation to reach the 18 mm. mark. All suggestions made by Friedlaender and other investigators were closely followed so that the technic was uniform.

Charts have been prepared in which 300 consecutive gynecologic patients are grouped according to the sedimentation rate, temperature, and leucocyte count.

TABLE I

SHOWING A COMPARISON OF THE RATES OF SEDIMENTATION, LEUCOCYTE COUNTS, AND TEMPERATURE IN GROUPS OF SIMPLE AND COMPLICATED FIBROIDS

| DIAGNOSIS | NO. CASES | SED. | TEMP. | HGB. | W. B. C. | POLYS |
|---|---|---|---|---|---|---|
| Simple fibroids | 26 | 76 | 98.6° | 72 | 7400 | 65 |
| Complicated with asthma | 1 | 36 | 98.2° | 80 | 8200 | 56 |
| Complicated with adnexal disease | 10 | 41 | 100.2° | 70 | 13320 | 73 |
| Complicated with pyelitis | 3 | 28 | 98.8° | 91 | 13500 | 65 |
| Complicated with chronic appendicitis | 6 | 43 | 98.6° | 68 | 6830 | 63 |

Chart I includes patients having a rapid sedimentation rate of less than thirty minutes associated with temperature and leucocytosis, and as a result we find that cases of postpartum infection, postabortal infection, pelvic abscess, wound infection and postpartum infection with peritonitis, fall within this limit.

Chart II includes cases with rapid sedimentation, a moderate leucocyte reaction, and a temperature of under 100°; here we find localized collections of confined purulent material,—as bartholinian abscess, pyelitis, and old pelvic inflammatory masses.

Carcinoma of the uterus has invariably shown a rapid sedimentation time with no corresponding rise in temperature or leucocytosis. In the differential diagnosis between tuboovarian inflammation and ectopic pregnancy, the sedimentation time has been rapid in tuboovarian inflammation and relatively slow, i.e., one hundred minutes or more, in ectopic pregnancy.

One of our most interesting observations has been in a series of 26 uncomplicated fibroids of the uterus in which the sedimentation time averaged seventy-six minutes, while in 10 cases of fibroid complicated with adnexal inflammation the time of sedimentation was only forty-one minutes, this notwithstanding that both temperature curve and white cell count showed no elevation above the normal. (Table I.) This rapid rate has also held true in fibroids complicated with asthma, and fibroids complicated by chronic appendicitis and pyelitis. These observations have definitely demonstrated the value of the test in showing the presence of infective processes whether confined to the pelvis or not. As a prognostic sign it also has a value when correlated with other clinical data as will be shown in Charts III and IV.

It is well established that foci of infection may remain quiescent for weeks, months or years, only to undergo exacerbation after opera-

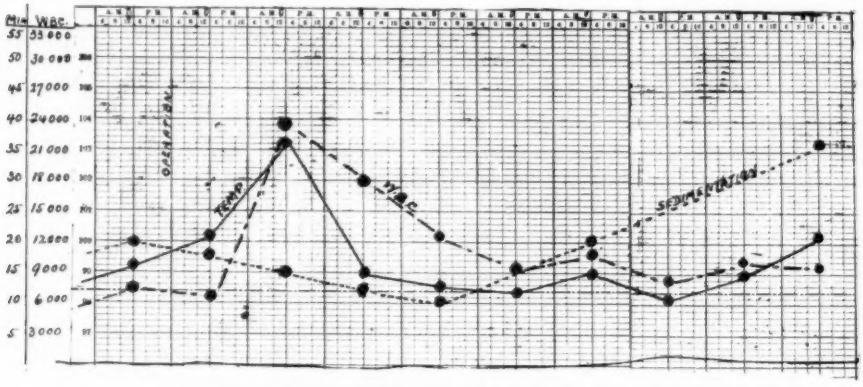


Chart IV.—Incomplete abortion with potential infection (low grade): This chart shows that the patient had a low grade temperature (99°) prior to operation. The leucocyte count was normal, but the sedimentation time was low (twenty minutes). The patient was curetted and on the sixth day following, her temperature rose to 103.2°, leucocyte count 21,800, with fifteen minutes sedimentation time. The prognosis according to these findings was not so good. The following day the temperature came down, the leucocytes were lower, but sedimentation remained about the same. In this case temperature and leucocyte count predicted a much more immediate prognosis than did the sedimentation time. The latter only began to show a gradual rise with the improvement of the patient.

tion, when they may produce peritonitis, parametritis, and blood-stream infections. Apparently the bacteria are buried in the tissues and are surrounded by a limiting wall of connective tissue. Trauma produces dissemination. Heretofore, the clinical history and Simpson's rule have been our only guides. The sedimentation test adds another safeguard. For example: in incomplete potentially septic abortions, appreciation of a rapid sedimentation time when associated with a normal temperature curve and low leucocyte count, has saved a number of women from having their uteri curetted and nature's barriers broken down. Its routine employment as a pre-

operative procedure in gynecologic cases will likewise safeguard the woman who is potentially infected, or warn us of her infectivity when the local barriers are broken down.

TABLE II

ANALYSIS OF THE AVERAGE SEDIMENTATION RATES, TEMPERATURES AND LEUCOCYTE COUNTS IN GROUPS OF ECTOPICS AND TUBOOVARIAN DISEASE

| DIAGNOSIS | NO. CASES | SED. | TEMP. | HGB. | W. B. C. | POLYS |
|---|---|---|---|---|---|---|
| Ectopics | 10 | 104.5 | 99° | 75 | 12,000 | 70 |
| Tuboovarian disease | 16 | 59 | 99° | 78 | 9390 | 66 |

We have found a low sedimentation time to be the earliest index of beginning postoperative peritonitis or parametritis. We would, therefore, conclude that in the sedimentation test, we have another aid in the diagnosis of infection and that when frequently repeated and correlated with the history, the temperature curve, and the white cell changes, it is a valuable index as to when to operate and a sign of prognostic value.

(*For discussion see page* 757.)

# THE RELATION OF BASAL METABOLISM TO STERILITY*

*(A Preliminary Report)*

By Jennings C. Litzenberg, M.D., Minneapolis, Minn.

IT HAS been known for years that marked disturbances of the function of the thyroid gland cause sterility, but the possibility that the milder alterations of that function may also affect fecundity has been given scant attention. As David Marine says: "The relation of the thyroid to the sex organs is the most ancient and classical interrelation of the functions of the glands of internal secretion, known to the ancients and a subject of daily gossip, it has passed down through the ages." With the hyperfunction of exophthalmic goiter or the hypofunction of myxedema, conception is rare. This preliminary report is chiefly upon a study of sterile woman with a basal metabolic rate only slightly below normal and showing none of the other usual symptoms of myxedema. The milder degrees of hyperthyroidism have not yet been studied.

We have approached the question from two angles: first, what proportion of sterile women have a low basal metabolic rate and can any of them be relieved by treatment, and second, what proportion of women with a low basal metabolic rate are sterile.

Our attention was first directed to this problem by one rather striking case, the principal features of which follow:

Mrs. B., aged twenty-six years, came to me, seeking relief from sterility. Her health had always been good. She was rather a large woman of unusual physical development. The most significant fact in her history was that her menses were always irregular, from three to nine times a year. Examination revealed only an acutely anteflexed uterus which was corrected by dilatation, not because we have much belief in its efficacy, but because we did not wish to neglect even the remotest possible cause of her sterility. For the same reason, although I have little faith in the clinical value of any of the ovarian extracts, she was given various preparations with the same result that followed the dilatation, namely, failure to conceive and no benefit to her menstruations. Before beginning any of our procedures the husband had been examined and found to be in good health; his semen was normal in every particular.

The patient had no symptom of myxedema, so we did not give her thyroid medication. Having failed in all the usual gynecologic procedures, I suggested a complete general physical examination in spite of the fact that she was the very picture of robust health. She said she would do anything that might make it possible for her to have a baby.

The only abnormality found was a basal metabolic rate of minus seventeen. Beginning in November, 1921, she was given five grains of thyroid extract daily.

*Presented at the Fifty-first Annual Meeting of the American Gynecological Society, Stockbridge, Mass., May 20, 21 and 22 ,1926.

On December 20, 1921, the basal metabolic rate had risen from minus seventeen to plus twenty-three. The dose was reduced and on January 17, 1922, her metabolic rate was normal, plus four. She was told to continue the reduced dosage but did not report until April 6, 1922, when she stated with much discouragement that she had not menstruated since January 5, but to her great joy and our surprise, examination revealed a three months' pregnancy. We did not dare, at that time, to believe that the treatment had anything to do with conception but rather considered it a coincidence. The treatment was continued throughout the pregnancy which terminated normally October 26, 1922.

In September, 1923, the patient came to the office saying that she wanted another baby but was unable to conceive and said that she would like to have her basal metabolic rate taken. The next day her rate was minus thirteen, so little below normal that we thought that her only pregnancy must have been only a coincidence, so far as our treatment was concerned. But, again, without expectation of success we gave her thyroid extract. On March 11, 1924, she came, not discouraged on account of amenorrhea but believing that she was pregnant because she had not menstruated since January 20, 1924. Our examination confirmed her conviction that she was pregnant, and her second baby was born October 26, 1924.

She took two grains of thyroid extract daily throughout this pregnancy and for a month after delivery, when her basal metabolic rate was normal, plus four. She continued to menstruate normally. We were now impressed with fact that two pregnancies had followed thyroid medication in the presence of a moderately low basal metabolic rate and we began our investigation by estimating the basal metabolic rate in other sterile women. But our enthusiasm received something of check when this same patient on December 1, 1925, came in and announced again that she was pregnant, not having menstruated since October 1. She had conceived without thyroid treatment, and we again feared, even though she had twice conceived under the treatment, that it was a coincidence and that her low metabolic rate was not a factor in her sterility, so we made a basal rate estimation the next day and we were a little more sanguine when we found the rate normal, plus eight. So our surmise that a normal rate is necessary to conception was still tenable. But we still wondered why the rate was normal when every previous determination was subnormal except when she was under treatment.

She knew of our quandary and asked if iodized salt which she had been using on her table for some time could account for her normal basal rate. Perhaps it did. The patient aborted December 17, 1925. She was told to discontinue the iodized salt for the purpose of testing its effect upon her basal metabolic rate. On February 7, 1926, her basal rate had dropped to minus three, and on April 27, 1926, to minus fourteen, so I believe that we may safely infer that her normal basal rate at the time of her third conception was in all probability due to the iodized salt.

Perhaps, then, it is not beyond reason to believe that the three pregnancies in this patient were due to the treatment which made her basal metabolic rate normal.

After this woman's second pregnancy we began to think seriously about the possibility of moderately low basal metabolic rates being in some cases a cause of sterility (or the index of a cause). Therefore, in those cases with a history and symptoms pointing to endocrine disturbances we determined the rate, and lately, in practically all sterility cases, the rate has been taken. Our results are presented as this preliminary report.

We have for analysis 69 consecutive cases of sterile women upon whom the basal metabolism rate has been determined. Of these, 44 came seeking relief for sterility; the remaining 25 were found during the course of a complete general physical examination to have a low basal rate and were sterile, but sterility was not the primary reason for their coming for examination. Of the 44 seeking relief from sterility, 22, or 50 per cent, had a basal metabolic rate of minus ten or below. We are following the usual rule of considering the normal rate to be between plus ten and minus ten.

Of these twenty-two, eight had a basal metabolic rate of between minus ten and fifteen; 5 between minus sixteen and minus twenty; 4 between minus twenty-one and twenty-five; 2 between minus twenty-six and thirty, and 1 minus thirty-seven. Of these twenty-two, eighteen received carefully supervised thyroid medication, of whom 6, or 33⅓ per cent, became pregnant within a short time after beginning treatment, usually within two months.

One patient, reported above, conceived three times, giving us a total of eight pregnancies, or 40 per cent, following treatment. None of these 6 women had other symptoms of myxedema, not even the woman with a basal metabolic rate of minus thirty-seven, and, excepting this one, none had a rate more than minus twenty, the highest being minus thirteen.

Having obtained these very suggestive results in women coming primarily on account of sterility, this question suggested itself: What proportion of women are sterile who come to the physician with other complaints than sterility for a general physical examination and are found to have a low basal metabolic rate?

In this group were 114 women with low basal metabolism rates, of whom 68 were married. Thirty-one, or 45 per cent, of these women were sterile, including 4 with symptoms of myxedema whom we would expect to be sterile. Omitting these 4, 27, or 39 per cent, were without signs of myxedema, yet they were sterile.

Weight is added to the probability of the low basal metabolism rate being the cause of the sterility by the fact that 40 per cent of the 114 women, both married and unmarried, had functional disturbances of menstruation.

In addition to 31 sterile women, there were 5 others who had no living children but had had one or more pregnancies resulting in abortion, miscarriage or stillbirth, two of the women later carried their babies to full term following treatment. So it appears that a normal basal metabolic rate is apparently not only necessary for conception to occur, but also probably necessary for a continuance of the pregnancy.

Definite conclusions cannot be drawn from this small number of cases, but there is a significance in our findings which demands fur-

ther investigation to establish the truth or falsity of the possibility that moderate or, as it has been called, "incipient" hypothyroidism may be a cause (or the index of a cause) of sterility. To this end we are instituting animal experimentation and are making this preliminary report with the hope that others may continue the study on women who are sterile, and in due time enough evidence may be accumulated to decide the question one way or the other.*

## SUMMARY

While the number of cases in this preliminary report are not sufficient to justify drawing final conclusions, the findings are significant because:

1. The relation between the thyroid gland and the ovary is well known.

2. Myxedema is certainly a cause of sterility.

3. Lesser degrees of hypothyroidism are, by the results of our investigation, apparently also a cause (or index of a cause) of sterility.

4. A normal basal metabolism rate is apparently necessary to conception and to a normal continuance of pregnancy.

5. Properly supervised thyroid medication will restore the basal metabolic rate to normal and in some cases result in conception.

6. Women who habitually abort should have their basal metabolic rate taken.

1009 NICOLLET AVENUE.

(*For discussion see page* 763.)

*I wish to acknowledge my indebtedness to my medical colleagues, Drs. Olga Hansen and James B. Carey, who made the general physical examinations and supervised the medication in all of our cases.

# THE INCIDENCE OF DENTAL CARIES IN PREGNANT WOMEN*

By Daniel E. Ziskin, D.D.S., Minneapolis, Minn.

*(Chief of Dental Staff, Minneapolis General Hospital, Assistant Professor, Department of Oral Surgery, University of Minnesota)*

MANY theories have been advanced as to the possible causes of dental caries in adult women. Among them, the belief that pregnancy plays an important rôle as a causative factor has become so widely accepted that it is almost universally regarded as a fact, both by the laity and by the professions.

It occurred to me, during my observation of the mouth conditions of hundreds of pregnant women coming to the prenatal clinic at the Minneapolis General Hospital, that it would be of interest to study the relation between pregnancy and caries. With this purpose in view, the present investigation was undertaken.

*In this study, only pregnancy was considered as a causative factor, while any other theory which may have a bearing on the cause of caries in pregnant women was excluded.*

It would seem logical to believe that if we examined routinely the mouths of pregnant women and noted the frequency of the occurrence of decayed teeth, and later compared this group with a control group of women in whom pregnancy never existed, the conclusions derived from this comparison would clearly indicate whether or not pregnancy, per se, is an etiologic factor in caries of the teeth. Particularly would this be true if we could find two groups of women in whom, all other factors being equal, pregnancy was the only difference.

The cases which we have statistically compiled were taken from the records of pregnant women attending the Minneapolis General Hospital Dental Clinic from 1922 to 1925. These cases number about 25 per cent of all cases attending the prenatal clinic. The mouth examinations were made, for the most part, during the latter months of the period of gestation. Some of the cases in the control group were obtained from the women attending the dental clinic at the Minneapolis General Hospital, and the others from the women applying for dental examinations at the College of Dentistry, University of Minnesota. Of the latter, forty-nine were married women and seventy-two unmarried. None of the control group were ever pregnant.

The only apparent difference in the two groups is pregnancy. The patients came to these two clinics for mouth examinations in search of dental caries, for mouth hygiene, and for x-rays. They were referred

*From the Department of Dentistry, Minneapolis General Hospital and the University of Minnesota College of Dentistry.

Read before the Twin Academy of Stomatology, March 9, 1925, and the Minneapolis District Dental Society, March 11, 1926.

TABLE I

CASE DISTRIBUTION FOR EACH AGE

| AGE | 15 | 16 | 17 | 18 | 19 | 20 | 21 | 22 | 23 | 24 | 25 | 26 | 27 | 28 | 29 | 30 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Pregnant Group | 1 | 7 | 8 | 25 | 36 | 47 | 51 | 50 | 38 | 40 | 44 | 36 | 22 | 22 | 15 | 20 |
| Control Group | 6 | 9 | 5 | 7 | 12 | 20 | 13 | 17 | 9 | 12 | 11 | 12 | 15 | 5 | 2 | 7 |

CASE DISTRIBUTION FOR EACH AGE—CONT'D

| AGE | 31 | 32 | 33 | 34 | 35 | 36 | 37 | 38 | 39 | 40 | 41 | 42 | 43 | 44 | 45 | TOTAL |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Pregnant Group | 17 | 21 | 17 | 15 | 13 | 10 | 15 | 7 | 6 | 5 | 1 | 3 | 3 | 1 | 3 | 599 |
| Control Group | 5 | 1 | 3 | 3 | 4 | 3 | 2 | 1 | 3 | 3 | 1 | 3 | 0 | 1 | 10 | 205 |

TABLE V

COMPARATIVE TABLE SHOWING AVERAGE FREQUENCY OF CARIOUS AND MISSING TEETH IN PREGNANT AND NEVER PREGNANT WOMEN

| AGE GROUPS | NUMBER OF CASES | | | AVERAGE AGE | | | CARIOUS | | | MISSING | | | CARIOUS AND MISSING | | | PER CENT CARIOUS | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | P* | H | C | P | H | C | P | H | C | P | H | C | P | H | C | P | H | C |
| 15-19 | 77 | 16 | 39 | 18.14 | 17.55 | 17.25 | 6.70 | 11.1 | 12.30 | 4.04 | 4.7 | 4.51 | 10.74 | 15.9 | 16.81 | 24 | 40 | 45 |
| 20-24 | 226 | 34 | 71 | 21.89 | 21.59 | 21.72 | 9.27 | 9.3 | 11.38 | 4.16 | 6.0 | 4.75 | 13.13 | 15.3 | 16.13 | 33 | 35 | 42 |
| 25-29 | 139 | 16 | 45 | 26.48 | 26.25 | 26.45 | 10.17 | 10.5 | 12.13 | 6.01 | 6.8 | 6.33 | 16.18 | 17.3 | 18.46 | 39 | 41 | 47 |
| 30-34 | 90 | 6 | 19 | 31.89 | 31.66 | 31.48 | 10.45 | 11.5 | 13.00 | 9.78 | 8.1 | 7.21 | 20.23 | 19.6 | 20.21 | 47 | 48 | 52 |
| 35-39 | 51 | 2 | 13 | 36.67 | 35.50 | 36.69 | 8.88 | 18.0 | 14.92 | 8.55 | 3.0 | 8.39 | 17.43 | 21.0 | 23.31 | 38 | 62 | 62 |
| 40-45 | 16 | — | 18 | 41.82 | — | 44.37 | 6.43 | — | 11.55 | 12.32 | — | 11.55 | 18.75 | — | 23.10 | 33 | — | 56 |
| Group | 599 | 74 | 205 | 25.95 | 23.00 | 27.05 | 9.22 | 10.4 | 11.67 | 6.01 | 6.0 | 5.94 | 15.23 | 16.4 | 17.61 | 35 | 40 | 43 |

*P=pregnant, H=hospital, C=control.

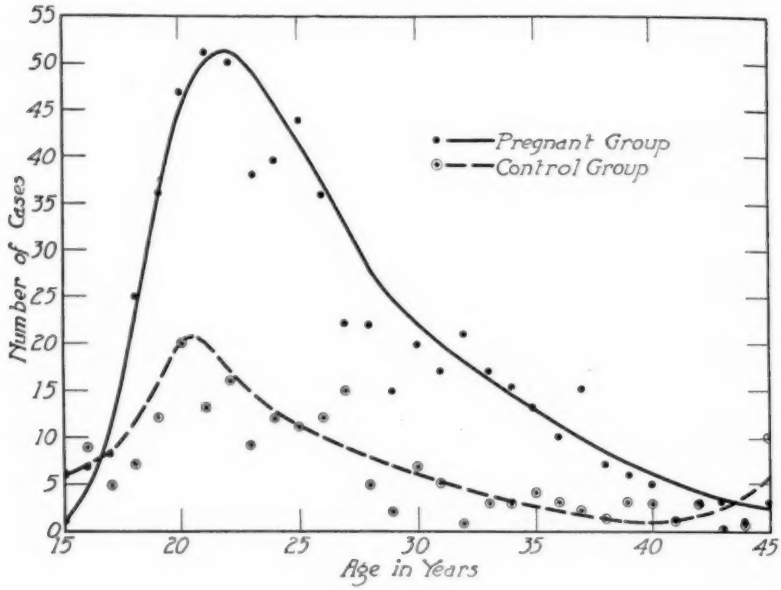
Number of Cases
Age in Years
Pregnant Group
Control Group

Fig. 1.

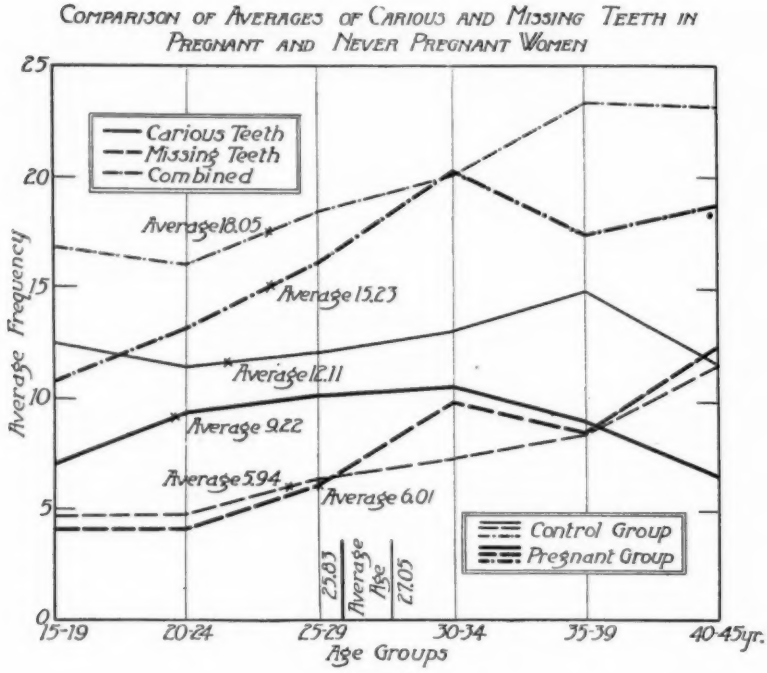
Comparison of Averages of Carious and Missing Teeth in Pregnant and Never Pregnant Women
Average Frequency
Age Groups
Carious Teeth
Missing Teeth
Combined
Average 18.05
Average 15.23
Average 12.11
Average 9.22
Average 5.94
Average 6.01
Control Group
Pregnant Group
Average Age
25.83
27.05
15-19
20-24
25-29
30-34
35-39
40-45yr.

Fig. 2.

either by physicians or by medical dispensaries for relief from some definite dental ailment.

*Thus, in this comparison, we have two groups of women, both attending dental clinics, both from the same social strata of life, both of comparable financial means, and both falling within the same age limitations.*

In considering the frequency of caries, every tooth filled, crowned, or decayed was counted. If a tooth had both a filling and a cavity, it was counted only as one occurrence and not as two. The tables, therefore, represent the number of carious teeth and not the number of cavities. I also counted missing teeth as an evidence of caries. Teeth may be missing from causes other than caries, yet, for the purpose of this classification, they were given similar consideration in both the pregnant and control groups, as it is reasonable to suppose that in both groups there would be a like number of teeth extracted for causes other than caries, and this study tends to show a comparison between the two groups rather than a record of tooth destruction.

The ages of the women in both groups range between fifteen and forty-five years, with the exception of six cases in the control group and two cases in the pregnant group which are beyond the age of forty-five. For convenience, the cases were separated into six age divisions as follows: fifteen to nineteen years; twenty to twenty-four years; twenty-five to twenty-nine years; thirty to thirty-four years; thirty-five to thirty-nine years; and forty to forty-five years. All cases beyond the age of forty-five were included in the last division.

Table I shows the distribution of cases according to the various ages in both groups. Here we find that over half the cases in both groups fall within a ten-year period between the ages of eighteen and twenty-eight. Fig. 1 is a smoothed curve, showing the data contained in Table I. The peak of this curve for the pregnant group is at age twenty-one, with 51 cases. For the control group, the peak is at age twenty, with 20 cases. There are, in all, 599 cases in the pregnant group and 205 cases in the control group. The cases compiled here are intended for a preliminary study. A subsequent report with a larger group of cases will be given later.

TABLE II

FREQUENCY OF CARIOUS AND MISSING TEETH IN PREGNANT WOMEN

| AGE GROUP | NUMBER OF CASES | AVERAGE | | | | PER CENT CARIOUS |
|---|---|---|---|---|---|---|
| | | AGE | CARIOUS | MISSING | CARIOUS AND MISSING | |
| 15-19 | 77 | 18.14 | 6.70 | 4.04 | 10.74 | 24 |
| 20-24 | 226 | 21.89 | 9.27 | 4.16 | 13.43 | 33 |
| 25-29 | 139 | 26.48 | 10.17 | 6.01 | 16.18 | 39 |
| 30-34 | 90 | 31.89 | 10.45 | 9.78 | 20.23 | 47 |
| 35-39 | 51 | 36.67 | 8.88 | 8.55 | 17.43 | 38 |
| 40-45 | 16 | 41.82 | 6.43 | 12.32 | 18.75 | 33 |
| Total | 599 | 25.83 | 9.22 | 6.01 | 15.23 | 35 |
| | | Group Averages | | | | |

Table II shows the cases in the pregnant group first separated into the six age divisions with the number of cases and average age for each. Next is shown the average incidence of carious and missing teeth for each division, first separately and then combined. The averages for the entire group are given below. The figures indicate teeth, carious and missing, in the average mouth for each age division and for the entire group, with the exception of the last column. Here is shown the per cent of caries only in teeth present at the time the examination was

TABLE III

FREQUENCY OF CARIOUS AND MISSING TEETH IN NEVER PREGNANT WOMEN

| AGE GROUP | NUMBER OF CASES | AVERAGE | | | | PER CENT CARIOUS |
|---|---|---|---|---|---|---|
| | | AGE | CARIOUS | MISSING | CARIOUS AND MISSING | |
| 15-19 | 39 | 17.25 | 12.30 | 4.51 | 16.81 | 45 |
| 20-24 | 71 | 21.72 | 11.38 | 4.75 | 16.13 | 42 |
| 25-29 | 45 | 26.45 | 12.13 | 6.33 | 18.46 | 47 |
| 30-34 | 19 | 31.48 | 13.09 | 7.21 | 20.21 | 52 |
| 35-39 | 13 | 36.69 | 14.92 | 8.39 | 23.31 | 62 |
| 40-45 | 18 | 44.37 | 11.55 | 11.55 | 23.10 | 56 |
| Total | 205 | 27.05 | 11.67 | 5.94 | 17.61 | 43 |
| | | Group Averages | | | | |

TABLE IV

COMPARISON OF CASES IN HOSPITAL AND DENTAL COLLEGE GROUPS

| AGE GROUP | NUMBER OF CASES | CARIOUS | | MISSING | | CARIOUS AND MISSING | | PER CENT CARIOUS |
|---|---|---|---|---|---|---|---|---|
| | | NUMBER | AVERAGE | NUMBER | AVERAGE | NUMBER | AVERAGE | |
| 15-19 | Dental 23 | 301 | 13.0 | 102 | 4.4 | 403 | 17.5 | 47 |
| | Hospital 16 | 179 | 11.1 | 76 | 4.7 | 255 | 15.9 | 40 |
| 20-24 | Dental 37 | 490 | 13.2 | 133 | 3.5 | 623 | 16.8 | 46 |
| | Hospital 34 | 318 | 9.3 | 205 | 6.0 | 523 | 15.3 | 35 |
| 25-29 | Dental 29 | 377 | 13.0 | 176 | 6.0 | 553 | 19.0 | 50 |
| | Hospital 16 | 169 | 10.5 | 109 | 6.8 | 278 | 17.3 | 41 |
| 30-34 | Dental 13 | 178 | 13.6 | 88 | 6.7 | 266 | 20.4 | 53 |
| | Hospital 6 | 69 | 11.5 | 49 | 8.1 | 118 | 19.6 | 48 |
| 35-39 | Dental 11 | 158 | 14.3 | 103 | 9.3 | 261 | 23.7 | 62 |
| | Hospital 2 | 36 | 18.0 | 6 | 3.0 | 42 | 21.0 | 62 |
| Total Number of Cases | Dental 113 | 1504 | 13.6 | 602 | 5.4 | 2106 | 19.1 | 51 |
| | Hosp'tal 74 | 771 | 10.4 | 445 | 6.0 | 1216 | 16.4 | 40 |

made. In the column headed, "Carious and Missing Teeth" it will be noted that there is a general tendency for a rise in the frequency of the occurrence of caries with an increase in age.

Table III shows a similar treatment of the control group. It will be seen that in the column headed "Carious and Missing Teeth" there is also a general tendency for an increase in the frequency of the occurrence of caries with an increase in age.

Since the cases in the control group were collected from two different sources, Table IV shows them separated according to each source and

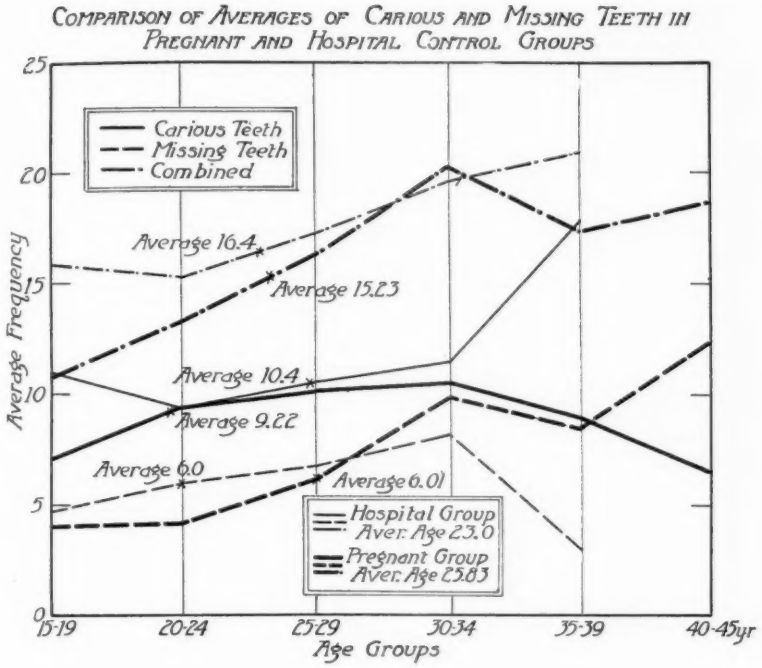
Comparison of Averages of Carious and Missing Teeth in Pregnant and Hospital Control Groups
Carious Teeth
Missing Teeth
Combined
Average 16.4
Average 15.23
Average 10.4
Average 9.22
Average 6.0
Average 6.01
Hospital Group Aver. Age 23.0
Pregnant Group Aver. Age 23.83
Average Frequency
25
20
15
10
5
0
15-19
20-24
25-29
30-34
35-39
40-45yr
Age Groups

Fig. 3.

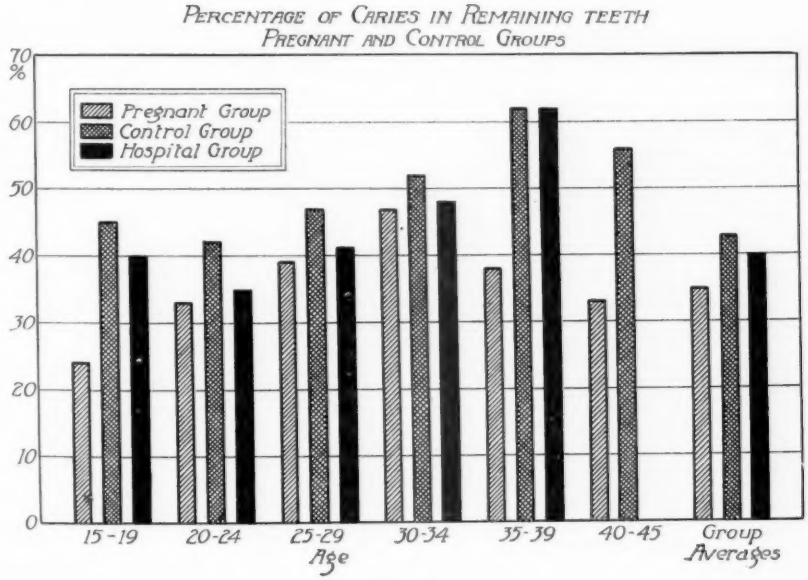
Percentage of Caries in Remaining Teeth
Pregnant and Control Groups
Pregnant Group
Control Group
Hospital Group
70
%
60
50
40
30
20
10
0
15-19
20-24
25-29
30-34
35-39
40-45
Group Averages
Age

Fig. 4.

a comparison made between them. They are classified as "dental," for the Dental College group, and "hospital," for the Hospital Dental Clinic group. The comparison was made to determine the difference in the incidence of caries in the two groups. Here, as before, we find a general rise in the frequency of the occurrence of caries as the age increases. The cases in the Dental College group show a greater incidence of caries than those in the Hospital group, but this can be explained by the difference in ages as is shown in Table V.

Table V is a combined table giving the comparison of the frequency of carious and missing teeth in the following groups: the pregnant group; the hospital control group; and the combined Hospital and Dental College control group. This table shows the incidence of carious and missing teeth in the control group as actually higher than those in the pregnant group. A comparison of the ages, however, reveals the fact that the average age in the control group is higher than in the pregnant group. This explains the higher incidence of caries in the control group as, from the accompanying tables, we concluded that there was a rise in the occurrence of caries with an increase in age.

TABLE VI

DISTRIBUTION OF PREGNANCY

| AGE GROUP | NUMBER OF CASES | PREGNANCY | | | | | | | | AVERAGE PREGNANCY | AVERAGE AGE |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | | 1st | 2nd | 3rd | 4th | 5th | 6th | 7th | 8th | | |
| 15-19 | 43 | 38 | 4 | 1 | — | — | — | — | — | 1.13 | 18.16 |
| 20-24 | 130 | 69 | 39 | 18 | 4 | — | — | — | — | 1.66 | 21.86 |
| 25-29 | 74 | 21 | 27 | 10 | 10 | 4 | — | 2 | — | 2.41 | 26.45 |
| 30-34 | 52 | 8 | 11 | 7 | 10 | 4 | 5 | 5 | 2 | 3.69 | 31.83 |
| 35-39 | 31 | 1 | 3 | 6 | 8 | 2 | 2 | 4 | 5 | 4.74 | 36.78 |
| 40-45 | 12 | — | — | 2 | 2 | — | 2 | 4 | 2 | 5.83 | 41.17 |
| Total | 342 | 137 | 84 | 44 | 34 | 10 | 9 | 15 | 9 | | |
| Average Age | | 22.07 | 25.21 | 27.57 | 31.11 | 31.00 | 35.33 | 35.43 | 37.00 | | |

Figs. 2 and 3 show, diagramatically, the data contained in Tables III, IV, and V. Fig. 2 shows a comparison, graphically, between the entire pregnant group and the entire control group.

Fig. 3 shows the comparison between the entire pregnant and only the hospital control group.

Fig. 4 is a bar diagram showing a comparison between the pregnant group, the hospital control group, and the combined control group. This study was made for the purpose of obtaining a comparison of the incidence of caries in remaining teeth only. That is, missing teeth were not counted. Here, as in the other figures, there is the same tendency to a rise in the frequency of occurrence of caries with an increase in age except in the latter division (forty to forty-five years) where there were so few cases that the comparison might well have been omitted.

A further consideration of the probability of pregnancy as a cause of caries may be shown in a classification of the pregnant women ac-

TABLE VII

COMPARISON OF THE AVERAGES OF CARIOUS AND MISSING TEETH FOR PREGNANCY ORDER AND AGE GROUPS (342 CASES)

| AGE GROUP | NO. OF CASES | AVERAGE AGE | FIRST PREGNANCY | | | SECOND PREGNANCY | | | THIRD PREGNANCY | | | FOURTH PREGNANCY | | | FIFTH PREGNANCY | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | C* | M | CM | C | M | CM | C | M | CM | C | M | CM | C | M | CM |
| 15-19 | 43 | 18.16 | 7.13 | 4.13 | 11.26 | 11.25 | 3.75 | 15.00 | 2.00 | 4.00 | 6.00 | | | | | | |
| 20-24 | 130 | 21.86 | 9.54 | 4.68 | 14.22 | 9.46 | 4.54 | 14.00 | 9.39 | 2.56 | 11.95 | 8.25 | 4.00 | 12.25 | | | |
| 25-29 | 74 | 26.45 | 9.33 | 9.05 | 18.38 | 10.52 | 5.41 | 15.93 | 12.80 | 3.40 | 16.20 | 9.50 | 9.50 | 19.00 | 16.75 | 2.25 | 19.00 |
| 30-34 | 52 | 31.83 | 9.75 | 8.37 | 18.12 | 10.27 | 8.09 | 18.36 | 13.71 | 8.00 | 21.71 | 11.70 | 10.50 | 22.20 | 11.50 | 12.00 | 23.50 |
| 35-39 | 31 | 36.78 | 12.00 | 6.00 | 18.00 | 12.33 | 12.66 | 25.00 | 12.00 | 6.50 | 18.50 | 9.13 | 9.39 | 18.51 | 8.00 | 10.50 | 18.50 |
| 40-45 | 12 | 41.17 | | | | | | | 15.00 | 8.00 | 23.00 | 8.00 | 14.00 | 22.00 | | | |
| Average | | 25.95 | 8.87 | 5.42 | 14.29 | 10.10 | 5.54 | 15.64 | 11.29 | 4.43 | 15.72 | 9.82 | 9.38 | 19.20 | 12.90 | 7.80 | 20.70 |
| Per cent carious | | | 33 | | | 38 | | | 41 | | | 43 | | | 53 | | |

COMPARISON OF THE AVERAGES OF CARIOUS AND MISSING TEETH FOR PREGNANCY ORDER AND AGE GROUPS (342 CASES)—CONT'D

| AGE GROUP | NO. OF CASES | AVERAGE AGE | SIXTH PREGNANCY | | | SEVENTH PREGNANCY | | | EIGHTH PREGNANCY | | | AGE GROUP AVERAGE | | | AVERAGE PREGNANCY | PER CENT CARIOUS |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | C | M | CM | C | M | CM | C | M | CM | C | M | CM | | |
| 15-19 | 43 | 18.16 | | | | | | | | | | 7.39 | 4.09 | 11.48 | 1.13 | 26 |
| 20-24 | 130 | 21.86 | | | | | | | | | | 9.45 | 4.32 | 13.77 | 1.66 | 34 |
| 25-29 | 74 | 26.45 | | | | 7.50 | 3.00 | 10.50 | | | | 10.61 | 6.48 | 17.09 | 2.41 | 42 |
| 30-34 | 52 | 31.83 | 12.60 | 6.60 | 19.20 | 12.40 | 9.00 | 21.40 | 9.00 | 14.00 | 23.00 | 11.50 | 9.05 | 20.55 | 3.69 | 50 |
| 35-39 | 31 | 36.78 | 15.00 | 8.00 | 23.00 | 9.25 | 7.25 | 16.50 | 13.80 | 9.20 | 23.00 | 8.93 | 7.23 | 16.16 | 4.74 | 36 |
| 40-45 | 12 | 41.17 | 2.00 | 19.50 | 21.50 | 6.00 | 8.00 | 14.00 | 6.00 | 11.50 | 17.50 | 7.17 | 11.50 | 18.67 | 5.83 | 35 |
| Average | | 25.95 | 10.78 | 9.78 | 20.56 | 9.55 | 7.47 | 17.00 | 11.00 | 10.77 | 21.77 | | | | 2.48 | |
| Per cent carious | | | 48 | | | 39 | | | 52 | | | | | | | 37 |

*C=carious, M=missing, CM=carious and missing.

cording to the number of pregnancies. If pregnancy is a cause of caries then the woman having the largest number of pregnancies should also have the largest frequency of carious and missing teeth. If, however, pregnancy is not a cause of caries, then the number of pregnancies will not add materially to the frequency of caries, but the age will be the determining factor.

Table VI shows the distribution of the cases in the pregnant group according to the six age divisions and to the number of pregnancies. Owing to the incomplete data, only those cases in which the number of

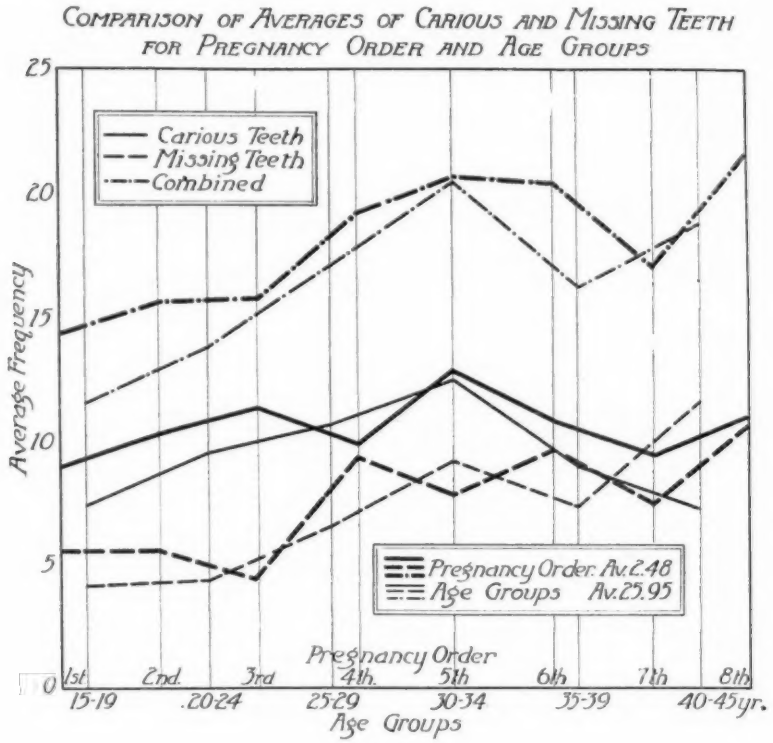


Fig. 5.

pregnancies were known were included in this table: 342 cases. Since there were so few cases of more than eight pregnancies, those exceeding this number were included in the eight-pregnancy group. This table also shows the average number of pregnancies for each age division as well as the average ages for both the six age divisions and the eight pregnancy groups.

Table VII shows the average frequency of carious and missing teeth in the known pregnancy group. The averages are calculated for each age division and for each pregnancy group. The per cent of carious teeth present at the time of examination is shown also.

Fig. 5 contains two sets of curves: one set showing average carious

and missing teeth according to pregnancy order, and the other showing average carious and missing teeth according to the age divisions; it also illustrates the data contained in Table VII. It will be noted here that the two curves rise in about the same degree. This tends to show that age is the determining factor in the increase in caries rather than pregnancy for if the pregnancy order influenced the frequency of caries, we would expect to find this curve much steeper.

### CONCLUSIONS

1. Pregnancy, *per se*, cannot be given as a cause of caries of the teeth.

2. The foregoing data show that there is a rise in the frequency of caries with an increase in age.

3. The number of pregnancies does not influence the frequency of caries of the teeth.

Appreciation is expressed to Drs. R. E. Scammon and F. L. Adair of Minneapolis, for their valuable suggestions in the compilation of these data.

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## THE FREQUENCY AND MEANING OF BACKACHE IN GYNECOLOGY*

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THE study of backache forms a most interesting problem in medicine. It is now definitely known that backache may arise from any number of conditions which may be coexistent in the same individual, therefore, in an analysis from an etiologic standpoint, it is necessary to proceed with the utmost caution. It has taken a long time for medicine to arrive at this conclusion, and there have been many steps in the development of the opinion.

When gynecology first developed as a specialty, it was believed that pelvic conditions were the chief cause of backache. The view changed only when it became known that relief did not often follow the procedures directed toward the pathologic conditions, and that men also were liable to backache. At that time little was known as to the etiology of lumbago or sciatica, or of the rôle the prostate plays in causing backache. When the pendulum swung to the other side, the gynecologists became most conservative in their statements relative to backache. Indeed, H. A. Kelly in the nineties constantly cautioned his students not to definitely promise relief from backache in the presence of pelvic inflammatory conditions or in the marked retroversions.

*Read at the Fifty-first Annual Meeting of the American Gynecological Society, Stockbridge, Mass., May 20, 1926.

The orthopedists made the next effort to establish the causes of backache. Meisenbach urged that since the sacroiliacs were true joints, they might slip when they became distended, and cause pain. In all, he reported 84 pathologic conditions of the joint. His work was followed by Lovett who, with Albee, also claimed that the sacroiliac articulations were true joints. They proved this by introducing colored fluids into them, thus giving outlines. However, the fact that the sacroiliac articulations were true joints had been known a long time. Albinius, in Leyden, nearly 300 years before, had proved it by convincing dissections. William Hunter had discovered the point anew and emphasized the importance of the joints in labor. A hundred years later, Senoir presented his specimens proving the point before the Paris Academy of Science. Luschka, in 1854, made similar observations regarding them and recorded them in the literature. The whole subject was most ably presented by Duncan's remarkable essays in 1867. Duncan emphasized the error of teaching that the pelvis is immobile since if it were true, men could walk in the most restricted manner and only after tremendous muscular exertion.

Lovett, however, is responsible for the first step in the modern conceptions of backache. He showed that it might be due to disease or displacements of the joint, to traumatism of the back, to arthritis of the spine as well as various pelvic conditions. However, he felt that the most common cause was static in origin and due to the overstrain of the muscles of the back, and that backache usually came from the irritation of these muscles, ligaments and fasciae. He also showed a large group of cases caused by strain or relaxation of the sacroiliac joint itself. Since the work of Lovett, the world has become well acquainted with the static factors. Nearly all believe that a forward displacement of the center of gravity is followed by undue strain on the back muscles. The pain results from fatigue and muscular irritation; although when the pain is in the neighborhood of the joint, it is difficult to say whether it comes from the muscles and fasciae about the sacroiliac joints or from the joints themselves. It is well known that in pregnancy the sacroiliacs as well as the symphysis are likely to have an exaggerated motility because of pelvic congestion. Because of the exaggerated stance in pregnancy, the sacrum may be readily pulled out of its place in articulation with the ilium and cause pain. Hence, any undue forward position of the body, if accompanied with a relaxed joint, would tend to displace the articulations and produce pain in that region. Thus, backache in fat men can be accounted for in many cases by muscular fatigue. The leg cramps in pregnancy doubtless follow muscular strain caused by Nature's effort to maintain a true footing in spite of the exaggerated abdominal position. With arthritis of the spine, there may be various nerve pressures; stiffness and lateral deviations of the spine may

also cause backache. There is x-ray evidence that osteophytes in the vertebrae and lipping in the vertebral edges may cause pain.

With the advent of focal infection, many other causes for backache were recognized. Billings stressed the importance of teeth, tonsils, sinuses of the head, the appendix, gall bladder, prostate, seminal vesicles and the female pelvic organs as chief sites of focal infection. The bacteriologic work of Rosenow and Dick has given this theory a reasonable amount of corroboration. It establishes the fact that backache may well represent some evidence of infection with or without disturbance of posture.

The influence of posture in gynecology was early urged by Dickinson. He called attention to the frequency of bad posture in women and proved his contention by a composite chart of the silhouettes of the back and abdomen of many cases. His work, however, has not aroused the interest that it merits. Since then, many men interested in gynecology have read papers on backache in women. Most of them discuss lightly the theory that gynecologic conditions may be an important factor in the complaint.

There are certain fundamental difficulties that attend any investigation of backache in gynecology. They attend the fact that bad posture is most common in women. Women do not stand as well ordinarily as men. They normally slouch and put overstrain on the posterior musculature. They have a high proportion of flat feet since they choose shoes that are governed by style and not by the needs of the wearer. Nearly all the complainants had borne children and had lax abdominal walls, and therefore tended to stand in an unusually ptotic position. The importance of fatigue is not sufficiently emphasized. Fatigue alone may be responsible for backache and because nearly all gynecologic cases complain of backache, it seems reasonable to assume that fatigue must be shared with static as well as with the pelvic conditions.

When reviewing this field, I found comparatively few articles that had been developed from a series of well controlled observations so that they could be regarded as definite conclusions. From the standpoint of pure theory, it seemed reasonable to believe that pelvic disorders might share with stance and fatigue the responsibility of causing backache, because women with comparatively slight pelvic pathology are likely to complain of backache when there is menstruation or premenstrual congestion. Women having backache usually say that it is more severe at the time of premenstrual congestion. There is also abundant proof that many women complain bitterly of backache until they are relieved by the cure of their dysmenorrhea, or of a marked uterine retroflexion, and that it ceases following the menopause. Backache in these cases is confined to the sacral or lower lumbar region and usually is referred to the upper sacrum. It is not

likely to be confused with the backache of fatigue which is usually referred to the dorsal region, although it is frequently confused with the orthopedic conditions.

Our interest in this subject was revived by reading the articles claiming that backache was rarely caused by gynecologic conditions. Up to that time, I had been impressed with the idea that many of my patients who complained bitterly of backache before operation were completely relieved after the correction of well-defined gynecologic conditions. I knew of no way to approach this subject but by a study of the pre- and postoperative symptoms.

Fortunately, we have a well ordered follow-up system in our Department in the University of California Hospital, in which we followed 90 per cent of cases with gynecologic conditions. Because we are conducting a study on the fate of the ovaries following hysterectomy, and of the meaning of retropositions, we had a large number of cases that had been followed carefully for several years, several hundred of them from four to nine years. We began our study with the review of 500 laparotomies which had been followed from one to eight years, many of which had vaginal work in addition. In calculating results, we thought that when the patient was permanently cured of backache by a gynecologic operation, we were justified in concluding that the backache was due to pelvic pathology, although there is always the chance that a good result might be due to an improved general condition. At the outset, we were fully aware of the many errors that might attend such an investigation, largely because the cases were not developed years ago primarily for such a study. Thus, to be of the greatest value, we should have a large series of cases which could be studied in similar groups of like postural defects of stance, etc., as long since advocated by Dickinson, or after they had been classified in various divisions, according to their individual reactions. Moreover, we should have as contrast a series of normal controls. While we could have arranged a series on this plan, it would have been too small to be of much value. For that reason, we confined our study of backache to groups of similar pelvic pathology.

As we proceeded with the work, we found that leads developed which required the addition of cases which only had cervical tears or vaginal relaxations. The results proved of such interest that we have now restudied the question from our histories of 1041 cases which have been followed accurately for a minimum of one and a maximum of eight years after operation.

Backache in the sacrum, or in the lower lumbar region, was a preoperative symptom in 49 per cent of 1041 gynecologic cases and did not constitute a complaint in 51 per cent. Our follow-up suggests that it was due to a gynecologic condition in 76.5 per cent of all who

had backache, since the complaint disappeared following operation and did not return again to constitute any but an occasional and comparatively slight symptom during the one to eight years follow-up period after operation, except in the presence of new gynecologic conditions. There were 23 per cent of the backache cases who were not relieved by the operation, composed of 16.5 per cent whose backache was not cured and 7 per cent whose backache was only improved after operation. We believe that these figures indicate that orthopedic conditions may have been responsible for at least 16.5 per cent of all the sacrolumbar backaches in the series and possibly part of the 7 per cent of cases whose backache was only improved following operation. There is, of course, the possibility that many of these cases might have had other etiologic factors, such as extremely bad tonsils or teeth which were treated in the operative routine. At any rate, the backache was diagnosed as orthopedic prior to operation in a very considerable number of cases in the series. We have included all such cases in the review because it is a study of the frequency and meaning of backache in gynecology.

Our series of 1041 cases consisted of 28 ovarian tumors, each tumor more than 8 cm. in diameter, 101 fibroids composed of cases in which fibroids were the major condition, 434 pelvic inflammatory disease cases, 290 retrodisplacements, most of them combined with descent, cervical injuries, and vaginal relaxations, 125 generally relaxed vaginal outlets of marked size including cervical lacerations, and 63 cases of complete prolapse. The cases were first studied in the above divisions. We made little headway. We presently found that injury and defects of the floor were a tremendous factor in the symptoms. We then arranged each of the various groupings accordingly as the pathology was entirely intraabdominal, or presented cervical pathology in addition, or combined cervical pathology and vaginal relaxation in addition to the intraabdominal condition, or was limited entirely to the cervical lacerations and vaginal conditions.

The vaginal relaxations were limited to those of large size in women in the childbearing age. The pelvic inflammatory cases consisted entirely of chronic cases since we do not admit the acute conditions. As our study progressed, it seemed reasonable to study the pelvic inflammations in two general groups, accordingly as they were mild or more severe cases. The milder pelvic inflammations consisted of 210 cases, in which at operation it was necessary to remove only one tube and ovary, or rarely both tubes and one ovary, with or without vaginal work in addition. Nearly all of this group presented posterior displacements of the uterus which were Nature's reaction to limit the inflammation. Such cases must be carefully distinguished from the noninflammatory type of displacement to which they bear no resemblance. The more severe type of pelvic inflammatory conditions

consisted of 224 cases in which it was necessary to perform a hysterectomy. We studied these in two groups, accordingly as the hysterectomy was complete or limited to the amputation of the uterine body. There were 83 panhysterectomies and 141 supravaginals. These divisions were subdivided again according to the treatment of adnexal disease and the condition of the vaginal floor.

Low backache was present in 15.4 per cent of the ovarian tumors; in 34 per cent of the fibroids; in 49 per cent of the pelvic inflammatory conditions; in 61 per cent of the retroversions and flexions; in 71 per cent of marked vaginal relaxations in the women in the menstrual age, and in only 22 per cent of the complete prolapsus cases. Of the cases in which backache was a preoperative symptom, the complaint was cured by the gynecologic operation in 50 per cent of the ovarian tumors; in 72 per cent of the pelvic inflammatory processes; in 79 per cent of the relaxed vaginal outlets; in 80 per cent of the fibroids; in 81 per cent of the retroversions and flexions and in only 37 per cent of the complete procidentias.

Various interesting features presented as the study developed. For a long time, they merely added to the confusion. First of all, we were greatly impressed with the infrequency of backache in ovarian tumors. Nearly all the ovarian tumors were of a size in excess of a five months' pregnancy, the largest one weighing 60 pounds. Backache was present once in 16 simple tumors; it was absent in an adherent pseudomucinous tumor of considerable size that had recurred after a previous operation. It was present in only two of six ovarian cancers, none of which had broken through the capsule. One of these tumors was of a size which entirely filled the abdomen. Backache was absent in one large retroperitoneal parovarian tumor.

The 101 fibroids represented many types. Severe backache was a complaint of one woman of 50 who had a goose egg sized tumor adherent in the pelvis and with almost complete calcification. Yet there were several cases without backache with a growth firmly fixed in the pelvis by adhesions, or with deeply fixed broad ligament and cervical fibroids. We found that backache was often absent even when the circulation was developing rapidly in quickly growing but otherwise uncomplicated tumors because there is, we believe, no chronic passive congestion. Backache was a rare complaint when there was a single large tumor, or when the patient had increased bleeding. Severe backache accompanied several tumors that were growing rapidly in retroflexions and in those that were developing degenerations. Backache was frequent when there were many small tumors and the uterus was in retroflexion. It also seems that backache was more frequent when the ovaries were adherent to the posterior wall. The fact that backache was present in but 15 per cent of ovarian tumors and 34 per cent of the fibroids deserves considerable attention. The back-

ache often disappeared when a rapidly growing uncomplicated tumor arose from the pelvis into the abdomen, reminding us of similar findings in retrodisplacements of a pregnant uterus temporarily incarcerated in the third or fourth month of pregnancy.

The importance of chronic pelvic congestion began to be appreciated when we studied the retroversions and flexions. Nearly all of these cases presented also more or less descent and cervical injuries and vaginal relaxations. All were, however, simple cases not complicated by pelvic inflammation. Our first great lead came after we separated the simple retroversioflexions from those which presented cervical or vaginal conditions in addition. We thus were able to study cases in which the grossly apparent pathology seemed at first to be limited to the retroflexion but which in reality deals with disturbance of the pelvic circulation. We are perfectly aware that there is not complete agreement that the so-called simple retroflexions and retroversions may ever cause symptoms. We have, however, proved to our complete satisfaction by a prolonged follow-up study of approximately 500 cases that chronic passive congestion in retroflexed uteri of the third degree may occasion definite symptoms under certain conditions. We believe that symptoms invariably result, when the uterus is enlarged, if the displacement has followed parametrial injuries during parturition. The symptoms accompanying retroversion disappear after the menopause, although there is a persistence of the anatomic condition. We feel that the following factors favor the development of symptoms in the simple retroflexions: enlargement of the uterus because of chronic passive congestion; varicose veins of the broad ligament; prolapsed enlarged ovaries which hang low in the pelvis. We have found no cases of simple retroflexions which present symptoms in the absence of these conditions. In another study, we attempted to see if symptoms finally develop in women who have moderately displaced uteri, not enlarged, who come in without symptoms. We felt that a certain number must return ultimately with more marked displacements and evidences of a disturbed circulation if the condition was acquired because of deep fascial injuries during parturition. We were forced to abandon the study because of the difficulty of keeping the entire series under continuous observation for a term of years in the absence of definite symptoms: although now and then a case returned after having been lost sight of for some years, coming back with a greatly enlarged uterus and complaining of symptoms. In the present study, we divided the simple uncomplicated displacements into various subdivisions, grouping them first according to the length of the uterus as shown at time of operation. This gave us much help in arriving at conclusions. Many of these cases had come in because of pain, or backache, or difficulty in menstruation. We found that no single factor appeared to be responsible

for backache. Thus, there were many greatly enlarged acutely displaced uteri without backache; many cases of prolapsed ovary without backache; many cases of varicosities of the broad ligament without backache, but no case was seen presenting backache without several of these conditions. Many of our displacement cases had bad tonsils or bad teeth without backache. Gallstones and a chronic appendix or gall bladder did not seem to be a factor in producing the condition. Cystitis usually caused a definite backache. Backache followed in a number of cases where the tubes were cut and ligated; we wondered whether because of ovarian adhesions.

An entirely different type of retroversioflexion is found in the cases of pelvic inflammation. In this group, the retrodisplacement is secondary and represents Nature's effort to wall off a pelvic infection. The uterus is usually not enlarged since the chief lesion is in the tubes or ovaries. Backache is not a frequent complaint in the latest stages of these infections since the pelvic circulation has been cut down after the infection has been limited. We operate only for the residues of the pelvic infection. Our study shows that backache was very frequent in chronic pelvic inflammatory cases when the uterus was enlarged and congested and there was frequent or severe bleeding. The backache was often most marked in the period of premenstrual congestion and was relieved after the case began bleeding. It is worthy of emphasis that backache was present in 61 per cent of the 290 noninflammatory retroversioflexions and in only 50 per cent of the retrodisplacements associated with tuboovarian inflammation. This we believe indicates the difference in frequency of chronic disturbances in the pelvic circulation. The backache was cured by gynecologic operation in 81 per cent of the noninflammatory retroversioflexions; in 87 per cent of the mild inflammatory conditions associated with enlarged adherent uteri in retroversioflexion, and in only 64 per cent of the later stages of the more extensive types of pelvic inflammation.

There seems to be a perfectly good reason for the long continuance of backache with the more severe pelvic conditions. The sacral plexus is separated from the pelvic viscera only by the pelvic fascia and readily receives its share of pelvic infection. It seems reasonable to believe that the nerve changes become chronic in the very bad cases with pelvic inflammation and require many months of rest before returning to the normal condition. We find that women are less disturbed with sacral pain after the menopause has developed, or operations or treatments have stopped or reduced the periodic pelvic congestion. We are strongly reminded of the backache that accompanies prostatic inflammation.

In the pelvic inflammatory group, we find points similar to those noted in the retroversions and flexions. The presence of a chronic gall bladder or gallstones or a chronic appendix did not seem to favor

backache. We are also greatly surprised to find a number of cases that came in for operation because of pain from former operative adhesions were free from backache. Cystitis again favored the production of backache. Two of the four cases of tuberculous peritonitis were free from backache; the backache was not cured in the other two cases. All of the group were treated by supravaginal hysterectomy with bilateral salpingo-oophorectomy.

We were greatly interested in the fact that none of six operable cases of cancer of the uterus had backache as a symptom. On the other hand, we rarely see cases with inoperable carcinoma in which backache is not an important symptom. Nor do we usually find backache in women whose complaint is idiopathic menorrhagia or the so-called fibroid uterus in which hemorrhage is a constant symptom.

A review of the relaxed vaginal outlets and endocervicitis cases gave interesting findings. There is no doubt but that backache follows congestion of an indurated parametrium caused by a lacerated cervix and some chronic infection. Backache was present in 71 per cent of women under forty years who presented very marked vaginal relaxations. It appeared as if it occurred more frequently when there were large high rectoceles or enteroceles than when there was large cystocele without cystitis. Traction on the broad ligaments may be responsible for the symptoms in which these cases resemble retroversioflexions with considerable descent and traction of the engorged parametrium. Backache was cured by gynecologic operation in 81 per cent of these cases of marked vaginal relaxation. Study of our tables shows that the percentage of backache is increased in any group of intraabdominal pelvic pathology if the cases have vaginal relaxations in addition.

In marked contrast to our findings in relaxed vaginal outlets are those in complete prolapse, a condition which, of course, is the last degree of vaginal relaxation. Backache in complete prolapse was present in only 22 per cent of cases and was cured by operation in but 37 per cent of them. This is opposed to the frequency of backache in 71 per cent of younger women with less marked relaxations of which 79 per cent were cured by operation.

The marked difference in the percentage of backache in relaxed vaginal outlet cases and in those of complete prolapse can be explained only on the basis of circulatory restrictions and loss of function in procidentia cases. Many of the prolapse cases stated that they had much backache when they were younger and before the uterus "came down." The large number of backaches in procidentia remaining uncured by operation calls attention to the fact that these women usually have markedly relaxed or pendulous fat abdominal walls, with bad posture, bad teeth, bad feet, and well-marked static conditions.

As the result of a preoperative and follow-up study of 1041 gynecologically operated cases that were carefully observed for periods varying between one and eight years, we believe we are justified in the following conclusions:

1. Sacral or sacrolumbar backache was a complaint in 49 per cent of 1041 women who came to gynecologic operation.

2. It constituted a complaint in 15 per cent of the 28 ovarian tumors; in 34 per cent of 101 fibroids; in 49 per cent of 434 pelvic inflammatory disease cases that came to abdominal operation; in 61 per cent of 290 retrodisplacements, most of which were combined with descent, cervical injuries and vaginal relaxations; in 71 per cent of the 125 marked vaginal relaxations in women under forty; and in only 22 per cent of the 63 complete prolapsus cases.

3. Backache may be ascribed to gynecologic pathology because it remained cured for periods ranging from one to eight years in 76.5 per cent of the 510 women of the series that had this as a preoperative symptom.

4. Backache was cured in the following percentages of the cases that had this preoperative symptom: 50 per cent of the ovarian tumors; 72 per cent of the pelvic chronic inflammations; 79 per cent of the relaxed vaginal outlets in women under forty; 80 per cent of the fibroids; 81 per cent of the retroversions and flexions; and 37 per cent of the complete procidentia.

5. Backache in gynecologic conditions is due chiefly to pelvic congestion. Comparatively slight defects in posture may favor the development of the condition.

6. Orthopedic conditions were responsible for between 16.5 per cent and 23.5 per cent of the total backaches of the series.

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(*For discussion see page* 759.)

# THE TECHNIC OF CESAREAN SECTION, WITH SPECIAL REFERENCE TO THE LOWER UTERINE SEGMENT INCISION*

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## THE UNSATISFACTORY NATURE OF THE UTERINE SCAR AFTER THE ORDINARY LONGITUDINAL INCISION

PRACTICALLY all writers have been forced to the conclusion that in a fair percentage of cases the scar after the ordinary longitudinal incision is not satisfactory. Couvelaire[1] states that in 17 per cent of cases there is an unsatisfactory cicatrix, in 10 per cent extreme thinning of the cicatrix, and in 2 per cent rupture of the uterus. Losee,[2] McPherson,[3] and Findlay[4] give somewhat similar experiences.

Eardley Holland[5] organized, early in 1920, among a large number of obstetric surgeons in Great Britain and Ireland, a follow-up inquiry into the subsequent obstetric history of hospital patients who had had cesarean section performed between the years 1912 and 1918, inclusive. He summarizes his results in the following table:

| | |
|---|---|
| Total number of cesarean section patients (excluding fatal and sterilized cases, and cases of repeated cesarean section where the first operation was performed prior to 1912) | 1,605 |
| Number followed up | 1,103 |
| Number in whom no subsequent pregnancy occurred | 616 |
| Number who subsequently became pregnant | 487 |
| Results of pregnancies: | |
| Delivery by natural passages | 78 |
| Repeated cesarean section | 352 |
| Abortion | 47 |
| Pregnant now | 86 |
| Rupture of scar | 18 |

These figures show that the frequency of rupture of the scar in subsequent pregnancy or labor (cases of abortion and early pregnancy excluded) is 18 in 448, or 4 per cent; also, that the proportion of ruptured scars to cases of delivery by the natural passages is 18 to 74, or 1 to 4.3.

Two of the most important contributions to the macroscopic and microscopic appearances of the uterine scar of the ordinary longitudinal incision are by Gamble[6] and McIntyre.[7] The latter found in his investigations that the scars contained a relatively large quantity

*Read, by title, at the Fifty-first Annual Meeting of the American Gynecological Society, Stockbridge, Mass., May 20-21, 1926.

of fibrous tissue. He did not find, even where the healing process was most satisfactory, an absence of fibrous tissue and a muscle regeneration, as Gamble states occurs under ideal conditions.

We take it, therefore, that there is definite evidence that the uterine scar after the longitudinal incision is not as sound a scar as is generally supposed, and that it frequently gives way completely or partially.

### THE REASONS WHY THE UTERINE SCAR WITH THE ORDINARY LONGITUDINAL INCISION IS SO FREQUENTLY DEFECTIVE

If a general surgeon were asked his opinion why the cesarean section wound is unsatisfactory in a number of cases, he would almost certainly give the answer that it is due to faulty technic. The explanation, however, is not so simple, for, as I shall try to indicate, the process of healing in the uterine wound is liable to certain disturbing factors which do not prevail in wounds elsewhere.

The first of these is the difficulty in securing complete asepsis. Now, this is specially difficult with the uterine wound, because of the danger of upward infection from the vagina. As cesarean section has often to be performed with the patient imperfectly prepared, and upon a structure so easily infected, it is not to be wondered at that even in the hands of the most careful and experienced operators infection of the uterus from below cannot always be prevented.

Another very important factor which militates against an absolutely normal healing in the uterine wound is the fact that the uterine muscle fibers during the puerperium are in a state of degeneration. An autolysis occurs in the muscle fibers. It is highly probable, therefore, that the healing process is interfered with in the early days of the puerperium as a result of this degeneration.

A third disturbing factor is the fact that the sheets of muscle which form the uterine wall are irregularly distributed, and this is seen very markedly in cesarean section whenever the uterus begins to retract. The surface of the wound, then, instead of being smooth, becomes irregular and puckered, and no matter how carefully the surgeon stitches the wound it is difficult to prevent the occurrence of small pockets of blood when he brings the surfaces of the wound together.

A fourth disturbing factor is the state of unrest of the uterus subsequent to operation. Not only does the uterus "retract," but from time to time it "contracts." If the uterus contracts before the sutures are inserted it will be observed that the edges and surfaces of the wound gape; while if the sutures are tied they appear strained when the uterus contracts. This alternate contraction and relaxation of the uterus, therefore, disturbs coaptation and lessens the hold the

stitches have on the tissues, and so favors the occurrence of small collections of blood between the coapted surfaces.

A fifth and very important factor is the necessity imposed upon the surgeon of using his ligatures not only as coaptors but as hemostatic agents. For the ideal healing of a wound, next to asepsis comes complete hemostasis. The general surgeon secures this by picking up bleeding vessels and if necessary applying ligatures to them. The obstetric surgeon cannot do this. He has to apply his sutures firmly if he wishes to stop bleeding and prevent the effusion of a certain amount of blood between the cut surfaces of the uterine wound.

There is yet another disturbing factor. If the placenta is situated on the anterior wall, and this occurs in about 40 per cent of cases, the operator will find that he has a layer of tissue peculiarly difficult to stitch and coapt exactly. It is very spongy, very friable, and contains large vessels; and, no matter how carefully he applies his sutures, blood collects between the edges and there is a tendency for a gutter to form along the internal line of the wound. Into this gutter at the subsequent pregnancy the membranes protrude and a hernia gradually develops: this is the ordinary method of rupture in a subsequent pregnancy or labor.

I maintain that these failures very decidedly militate against an absolutely sound uterine cicatrix.

### ADVANTAGES OF THE LOWER UTERINE SEGMENT INCISION

Anyone who has employed this method and seriously studied the formation and anatomy of the lower segment must be impressed by the following advantages this area presents to the surgeon who is anxious to secure a sound uterine cicatrix. Both Eardley Holland and I have already referred to this matter in our writings on the subject.

1. The wall of the uterus in this area is thin, especially if labor has been in progress for some time; it is often not more than one-sixth of an inch in thickness.

2. The tissue consists of fibromuscular tissue as in the upper segment, but here the fibrous tissue is much more abundant. McIntyre has tried to estimate the relative proportion of muscular and fibrous tissue.

3. It is less vascular. It is surprising how slight is the bleeding when an incision is made in this area.

4. As a result the surfaces of the wound can be more accurately approximated, and the formation of these pockets of blood clot already referred to can be prevented.

5. This area of the uterus, although it does not remain absolutely

inactive after the uterus is emptied, is more passive; and the wound has a chance of healing better, for it is less disturbed than is a wound in the upper segment. One has only to look at the appearance of the lower segment, as shown by frozen sections, to appreciate this point and its importance.

6. The wound is completely covered with the bladder wall and peritoneum. This keeps the wound extraperitoneal, limits slight infection, and prevents any adhesions of the uterus to surrounding tissues and structures.

7. Should infection unfortunately occur, it is an area that can be reached from the vagina should that be thought necessary, for the cervix can be pulled down, the bladder reflected from the cervix, and the wound drained.

8. The wound in the lower segment is not put on the stretch during a subsequent pregnancy: it is only after prolonged labor that this occurs. Very different are the conditions in the upper segment when during the whole time of pregnancy one is in doubt as to how the scar will stand the strain of the ever-increasing distension of the uterus and the active contractions of labor.

9. As far as can be judged from the literature, the few examples of weakening or rupture of the scar with the lower segment incision have been cases in which a longitudinal incision was employed. The objections to this incision are detailed under "Technic."

## TECHNIC OF OPERATION

The patient is placed in the Trendelenburg position. One-half c.c. of pituitrin is injected into the triceps muscle to secure prompt contraction when the uterus is emptied. A longitudinal incision, about six inches in length, is made through the abdominal wall in the middle line, the lower end just reaching the symphysis pubis. The lower end of the wound is then retracted over a protecting layer of gauze with a Doyen's retractor. The rest of the abdominal cavity is protected by packing off the upper portion of the operation area with gauze. A transverse incision is made through the loose peritoneal covering of the uterus, about halfway down the lower uterine segment. In recent cases my assistant, Dr. Hendry, and I have been employing a curved transverse incision with the convexity directed downwards. The object of this line of incision is to lessen the risk of injuring the vessels at the side. We are convinced that it is a much safer incision than the longitudinal one, for with the latter there is great risk of the wound extending into the upper contractile portion of the uterus. It is an interesting fact that in the few cases of rupture or weakening of the scar of the lower uterine segment incision, the line of incision in all the cases has been vertical. If the head is not

easily accessible, pressure on the fundus through the abdominal wall often brings the occiput into the wound. To avoid handling, especially in presumably infected cases, the head may then be picked up with a pair of short obstetric forceps, used as guides and not as tractors, and the delivery completed by pressure on the fundus. The umbilical cord is ligated and divided in the usual way. A self-retaining retractor is now inserted to keep the sides of the abdominal wall apart. If the cervix is not well dilated, the placenta is delivered by compression of the fundus through the abdominal wall, and traction on the cord. An intramuscular injection of a sterile preparation of ergot is given at this stage. If the cervix is known to be well dilated,—and this is a most important advantage in an infected case,—the placental end of the cord is dropped back into the uterus, the placenta and membranes being expressed *per vias naturales* when the abdomen has been closed. By this means intrauterine manipulations are reduced to a minimum. The edges of the uterine wound are now picked up with fine tissue forceps or temporary silk ligatures. The mucous membrane, with the innermost portion of the muscle coat, is now sutured with a continuous No. 1 chromic catgut suture, the edges of the mucous membrane being directed inwards towards the uterine cavity. The remainder of the muscular coat is then carefully approximated and sutured with a continuous No. 2 or No. 3 chromic catgut suture. Great care must be taken to secure completely the lateral extremities of the incision. The peritoneum over the uterus is then closed with a continuous No. 1 or No. 0 catgut suture, the operation area being thus completely shut off. The gauze packing is now removed, and the abdominal wall carefully closed in layers. The surface of the wound is secured with an anchored dressing.

### RESULTS OF FIRST AND REPEATED OPERATIONS

The following is a brief summary of results in a series of 107 cases:

| | TOTAL | MATERNAL MORTALITY | PERCENTAGE |
|---|---|---|---|
| Clean cases | 82 | 0 | 0 |
| Doubtful cases | 25 | 4 | 16 |

In "clean" cases are included only those in which all preoperative vaginal examinations were carried out in hospital; "doubtful" include all the others. The interference in the latter series varied from the previous unsuccessful application of forceps under domestic conditions to unsupervised vaginal examinations by nurses or midwives: in twelve of the series, including two of the fatal cases, not less than five such examinations had been made in each case.

RECORD OF SUBSEQUENT LABORS IN 26 OF THE ABOVE CASES

| | |
|---|---|
| Second cesarean section of the lower uterine segment type | 17 |
| Second cesarean section of the classical type | 6 |
| Spontaneous delivery of a smaller child | 2 |
| Low forceps | 1 |

There were no maternal deaths in this series. In only one of the cases already referred to was any thinning of the scar found at the second operation, and that in a case where the patient had been several hours in labor.

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## THE OCCIPITOPOSTERIOR POSITION*

By Charles S. Barnes, M.D., Philadelphia, Pa.

NO APOLOGY is offered for presenting this much discussed perennial subject. The condition, the most common anomaly of pregnancy and labor, is of much importance and demands to be kept before the profession until we much better understand how to cope with it.

In regard to its frequency, as a primary condition, there is a wide variance of opinion. The percentages given for this position range from 11 per cent in a series in Sloane Hospital, 17 per cent in Johns Hopkins, 26 per cent in a series of Dubois, to an estimate of 30 per cent right occipitoposterior by Shears. Potter states that he finds it in 60 to 70 per cent of his cases, because he examines them early, before rotation has occurred. His protagonists and disciples will perhaps credit him with correct observation, while his antagonists will charitably say that he is deceived. Quibbling over its exact frequency is not profitable, but it is an important fact that this is by far the most common anomaly in obstetrics. Breech presentation, the next most common one, is only 3 or 4 per cent. It seems a fair average estimate that 20 to 25 per cent of all cases of labor begin primarily as occipitoposterior.

The condition is vexing, perplexing, often most troublesome and difficult, and not infrequently results seriously—even disastrously. It is responsible for a high fetal mortality and for frequent maternal morbidity. Cragin reports a series of 20,000 cases of labor, with 13 per cent persistent occipitoposterior positions, showing the high fetal mortality of 7.66 per cent. Hirst places it at more than 9 per cent. DeLee says of it, "The condition itself and the operations performed by reason of it, cause untold and untellable suffering; the chil-

*Read at a meeting of the Philadelphia Obstetrical Society, January 7, 1926.

dren's brains are damaged, the mothers' soft parts lacerated and destroyed. I am convinced that, in the United States, ten times as many babies are lost from this complication as from contracted pelvis." It is worthy of note, however, that contracted pelvis of minor or moderate degree is not infrequently a coincident condition or a causative factor in the anomaly.

The condition is often carelessly or ignorantly overlooked, not diagnosed, and labor blindly allowed to drag on, with little progress, until, in desperation, the attendant is obliged to do something. Early positive diagnosis is not generally easy; the multiplicity of methods of management suggested are confusing. I hope to emphasize some important points of diagnosis (diagnosis being so essential to proper treatment), to emphasize some generally approved principles of management, and also possibly to present a little which is more or less new, or not generally comprehended or accepted.

Doubtless a very large proportion of these cases have never been positively diagnosed as to whether primarily anterior or posterior, and clinically, by good fortune, it made little difference. Therefore, commonly, alert, intelligent, watchful, active expectancy is the treatment, provided the obstetrician has eliminated in his study of the case, certain causes of the anomaly; e.g., an undue disproportion (a markedly contracted pelvis, or an oversized child), or a tumor or other condition obstructing the birth canal. In such case, elective cesarean section should have anticipated labor. Or if placenta previa be present, perhaps an etiologic factor, the treatment of the latter encompasses the former.

Few occipitoposterior relations occur on the left, but many on the right. If the fetal back is found on the mother's right, it is generally reasonable to assume a probable diagnosis of "right occipitoposterior" rather than "anterior" until proved otherwise. On the right side, the posterior relation is perhaps twice as common as the anterior. A slightly contracted pelvis, especially in a primipara, is another factor adding weight to the probable diagnosis; or some other departure from the normal, sometimes slight, such as relaxed maternal tissues. The signs of this condition determined by external examination include asymmetry of the abdominal ovoid, sometimes a depression above the pelvic brim anteriorly, opposite to the fetal back, the latter toward the maternal dorsum; fetal small parts in relation to the mother's anterior abdominal wall; fetal heart sounds in the mother's flank. Beware, however, of the latter, because, as a result of the frequently accompanying deflexion of the head of the fetus, cardiac sounds may be best transmitted through its anterior chest wall, which is pushed out against the ventral maternal tissues, thus leading to the false belief of an anterior position. The plain fact is that positive diagnosis of the condition must often be held in abeyance until dilatation of the cervix is sufficient

to admit several fingers for palpation of the vertex. The common dictum that the small fontanel (the meeting point of three sutures) is in the posterior part of the pelvis and the anterior fontanel (the meeting place of four sutures) in the anterior part of the pelvis is correct; but practically, it is not always easy to determine which is which. Palpating an ear, especially the meatus and noting the direction of the larger, freer portion of the auricle, there need be no question about the latter being directed, in these cases, posteriorly. A positive diagnosis can thus be made.

Because of lack of nice adjustment between the fetal head and the lower uterine segment, the patients in all cases of occipitoposterior position are prone to have insufficient and irregular labor pains; dilatation of the cervix is apt to be slow, and early rupture of the membranes is frequent,—true in all anomalous relations of fetus to mother.

If dilatation is finally completed, the mechanism of the second stage, though frequently, as stated, accomplished spontaneously, is usually also prolonged. The wider part of the fetal cranium, the occiput, is seeking to engage in the narrower part of the available space of the pelvis; that is, the biparietal diameter in relation to the narrower posterior portion of the pelvis, in contrast to the bitemporal in relation to the roomy anterior portion. This relation causes increased resistance, impedes progress and tends to extend the fetal head, bringing larger circumferences of the fetal head in relation to the birth canal. Therefore engagement and descent are usually slow. Rotation, if it occurs, must be accomplished through an arc of 135 degrees, instead of the usual 45 degrees—three times the distance required in anterior positions. These cases, therefore, at best, are usually much prolonged and demand alertness, good judgment, and skill in their management.

A patient in late pregnancy with a probable diagnosis of occipitoposterior, should be instructed that, when reclining, she should lie on that side related to the fetal back; so during labor. Authorities claim that the change from a posterior to an anterior position has resulted from such attitude of the mother. The knee-chest position is also advised, or the less uncomfortable lateral prone with the hips elevated. Purely external manipulation is usually futile.

The obstetrician in attendance upon the case with a probable diagnosis of occipitoposterior seeks to lessen as far as possible, the suffering and the exhaustion frequently incident to the first stage, by approved general and often by special measures, such as the Gwathmey method. If labor seems to be progressing favorably, he may avoid vaginal examination, until such time as he believes the cervix to be completely or nearly dilated. A rectal examination may meanwhile be helpful.

If progress seems to be unsatisfactory, early vaginal examination is indicated, the attendant being prepared at this time, especially where

premature rupture of the membranes has occurred, to introduce, if need be, a hydrostatic bag, as an aid to complete dilatation.

Happy is the attendant who finds the cervix completely dilated or favorable for manual completion. This examination, sometimes the first internal one, should be thorough (the patient anesthetized), the examining hand in the birth canal palpating the head, noting its relation, whether above the inlet, engaged, or well in the pelvic cavity; whether fixed or freely movable, and judgment formed as to the relative size of the head and the pelvis. Even at this stage of labor, the examiner may find (it should be seldom so late) that he has been deceived as to the degree of pelvic contraction or size of the fetus and that cesarean section is indicated.

Usually, in the second stage of labor, one of a few safe practical courses is available. If the mother and child are in good condition, if the head is descending, and especially if anterior rotation of the occiput has already begun, this step in mechanism may be aided, at the height of a pain, by increasing flexion of the head (pushing up the forehead, Hodge's maneuver, or drawing down the occiput). Also by pushing back the sinciput or drawing the occiput forward, rotation thus encouraged, may be spontaneously completed. This accomplished, delivery by the natural powers may ensue, or if not, when the occiput has advanced to the anterior quadrant of the pelvis, the forceps may complete delivery just as in a primary anterior position.

While, to repeat, a large proportion of such cases either end spontaneously or may readily be terminated by the aid of forceps, as just indicated, yet perhaps one or more out of five such cases persistently and obstinately remain with the occiput directed toward the sacroiliac joint or rotate toward the sacral cavity.

Probably at least 5 per cent of all labors (the estimate is as high as 13 per cent) are *persistent* occipitoposterior. J. Whitridge Williams reports 5488 cases of labor with a proportion of 11 per cent. Shears estimates 10 per cent or more. So few of these terminate spontaneously, face to pubes (I have never observed more than two or three), that some artificial aid must be employed. A very large proportion of these cases demand artificial aid in the *upper part* of the pelvis, or, not infrequently, before there is engagement or even fixation of the head in the pelvis. This is especially true of primiparas. Uterine contractions, perhaps at no time good, and voluntary efforts, both become weaker, the patient is irritable and has signs of approaching exhaustion, often before or by the time the cervix is dilated.

The latter completed, a confidence may be felt that, very generally, delivery can promptly and safely be accomplished in one of two ways; either by version and extraction, or by manual rotation of the fetus followed with forceps. I approach these cases with an open mind, prepared to do whichever in my judgment is safest and best. As a preliminary, the lower birth canal is freely stretched from the vaginal

vault downward, the outlet receiving special attention. Usually there is no necessity for undue haste in the process of delivery. If not previously done, the relation of the child to the maternal structures is studied, as outlined above. If the head is freely movable above the inlet or, though apparently fixed, can, under anesthesia, be readily pushed up, podalic version and breech extraction is elected. Williams says: "When the head is arrested at the superior strait, version is resorted to as soon as one is convinced that spontaneous advance will not occur." If the head is engaged or low in the pelvis, manual rotation followed by forceps extraction is the choice. It is exceedingly rare that one of these procedures cannot be carried out. Failing in these, forceps delivery with the occiput posterior is usually quite possible. Whatever the method of delivery, especially if the latter, a mediolateral perineotomy is often clearly advisable. Delivery is thus facilitated and a deep perineal laceration avoided.

Emphasis of some points in the manual rotation of the fetus seems important. For example right occipitoposterior is the common condition. The patient in the lithotomy position, the maneuver will be facilitated if the operator takes a relatively low position in relation to the birth canal. Introducing his left hand and everting it so as to grasp the occiput of the child, the fingers at least as far forward as the right fetal ear, he gradually begins rotation of the occiput forward around the right arc of the pelvis. Coincidently, the mother being well relaxed by anesthesia, the external hand manipulates the chest of the child, stroking the anterior shoulder towards the left of the mother. An assistant may help in the latter procedure. Gradually, cautiously, the occiput is brought into the right anterior pelvic quadrant, may be near to the symphysis. This rotation is very generally readily accomplished. Formerly I (sometimes to my great disappointment) failed at this crucial point, namely, holding the head in the corrected position while applying the forceps. The common teaching and practice has been to apply the left forceps blade first, for no sufficient reason except that the forceps is so made as to lock more readily when the blades are applied in this order. Recently this order of application in corrected right posterior position has been reversed. The left hand, having rotated the head, is kept in place to hold it and to guide the right blade to the right side of the fetal head. The latter applied, effectually holds the head in the corrected position. The left blade is then easily introduced, and while the process of locking is a little awkward, yet it is readily done by shifting the handles. This accomplished, the forceps delivery becomes one of right occipitoanterior. DeLee practices and advocates grasping the occiput with a vulsellum to fix it until the forceps is applied, but I think the method I employ is better and simpler. It was my purpose to have a forceps made for use in such cases, no different from the ordinary modified Simpson forceps, except designed to lock the reverse of the common one, the right blade to be introduced first. On inquiry, it

was learned that a member of our Society, Dr. Frederick E. Keller, had recently devised such an instrument.

The remaining method of delivery deserves, I believe, only brief discussion. This is commonly known as the Scanzoni method or maneuver. It consists, in principle, of application of forceps to the sides of the child's head (often most difficult), of rotation of the head so grasped, until the forceps is "upside down," then of removal of the forceps, and a reapplication, such an application as would be done after manual rotation. Special forceps have been advocated. Some authorities claim excellent success in the procedure, e.g., Williams, Shears, R. C. Norris, and Edgar. Many English authorities condemn the procedure; the German and French are said to take a more liberal view of it. It has warm advocates and violent opponents. However, all agree as to the danger involved to the child and to the mother; and, almost universally, advocates of the method, as well as opponents, emphasize the belief that the procedure is unsafe except in the hands of an expert. Why then practice or attempt a plan generally recognized as one beset with grave risk, when the simple safe procedure of manual rotation is readily available and may be made almost universally successful? DeLee and others have discarded the Scanzoni maneuver, and the former has not found it necessary to deliver with the occiput posterior, other simpler means proving more successful.

## CONCLUSIONS

Many physicians fail either to comprehend the frequency of this anomaly or to appreciate that such labors are always more or less complicated, often most seriously so.

Diagnosis is too seldom made, and when made, so often too late for an intelligent and successful conduct of the case. The case frequently drags on, the attendant in careless ignorance of the condition.

A tentative working diagnosis may generally be made late in pregnancy or early in labor. If not sooner, certainly by the end of the first stage, a positive diagnosis should be made by palpating the fontanelles and particularly an ear.

The first stage, often much prolonged, should be so managed as to minimize the suffering and the exhaustion menacing the mother.

The first stage completed, the second may often, with great advantage to both patients, be shortened. Unless there is good evidence of proper spontaneous progress, active intervention, either with podalic version and extraction, or with bimanual rotation and forceps extraction, should be practiced. If the latter procedure is elected, it is indicated to introduce that blade of the forceps first which corresponds to the side of the pelvis to which the occiput is related. Thorough manual dilatation of the lower birth canal should precede either of the above plans of procedure.

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(*For discussion see page* 771.)

# FURTHER STUDIES IN SEDIMENTATION*

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SINCE our first publication (Baer and Reis[1]) there has been no new theory advanced which satisfactorily explains the phenomena of the sedimentation of erythrocytes. In 1924-5, Mandelstamm[2] found that, although the sera of pregnant women and the sera of infectious cases show an increase in their ability to agglutinate many bacteria, that this agglutination could in no way be connected or identified with the sedimentation reaction. Sometime earlier this same investigator[3] definitely proved that the sedimentation rate is dependent upon some change in the properties of the erythrocytes themselves as well as changes in the plasma content.

Salomon, *et al*,[4] attempted to show a relationship between the cholesterol content of the serum and the sedimentation rate but were unable to do so. Kaufman[5] finds a lowered concentration of the blood plasma in the sedimented blood as compared with the unsedimented, and Groedel and Hubert[6] find no parallelism between the speed of sedimentation and the viscosity of the serum. Opitz and Frei[7] find no connection between the specific gravity of the plasma or the erythrocytes and the sedimentation rate.

The most rational explanation and that which is most generally accepted is based upon the number of negative charges carried by the erythrocytes. As the negative charges are taken off, either by the positively charged agglutinins present in the blood (Clausser,[8] Fahraeus,[9] Hueber,[10] Vorschuetz,[11] Nees,[12] Schurer and Einer[13]), or by the globulins (whose presence increases the viscosity and decreased the negative charges, Salomon,[14] Vorschuetz,[11] v. Oettingen,[15]) the erythrocytes no longer repel each other and tend toward an increased rouleau formation. The speed with which they sink is directly proportional to the massiveness of this rouleau formation.

In the technic, the Linzenmeier hard glass tubes, 6.5 centimeters long and 5 millimeters in diameter, have now been generally adopted. They are scaled at the 1 c.c. level and also at the 6, 12, 18 and 24 millimeter levels† (Baer and Reis[1]). In this report, readings were based solely upon the speed with which the sedimenting erythrocytes reached the 18 millimeter mark. It was found unnecessary to use the

*Presented at the Fifty-first Annual Meeting of the American Gynecological Society, Stockbridge, Mass., May 20, 21, and 22, 1926.

†These tubes were obtained from G. A. Ingram Co., 202 Bagley St., Detroit, Mich.

reading of levels attained at arbitrary time intervals such as one hour, etc.

One time-saving improvement has been devised by Haenkel and proved reliable by Herold,[16] who examined 200 cases, using both the Linzenmeier and the Haenkel technics. The latter is a centrifuge method which has the advantage of requiring only five minutes of centrifuging before the readings may be made, but require standardizing.

The sedimentation test in gynecology has been on trial for some years in this country and abroad. Its value has been emphasized by the following investigators since the appearance of the last article by the authors[17]; Popper and Kreindler[18] find the test a valuable aid in diagnosis and prognosis and with the simplicity of the technic deserves daily use; Nitschman[19] uses the test in the differential diagnosis of uncertain cases, especially in conjunction with a complete blood count; Frosch[20] believes that the sedimentation test is more delicate than the blood count can be; Nees[11] recommends it in all infectious cases; Bochner and Wassing[21] believe that it is "not only an index of the degree of absorption of catabolic products but also an index of the degree of resistance of the host and the virulence of the invading organism." Herold[16] uses the test routinely. Three workers report unsatisfactory results, Cherry,[22] Neuman,[23] and Schmitz.[24] Neuman states that in "the gynecologic cases which offer difficulties in determining the presence of inflammation, the results of the sedimentation test will not definitely decide the question," yet gives figures which are roughly comparable to the results usually obtained. His difficulty would seem to be one of interpretation. The figures obtained by Cherry and by Schmitz and Schmitz, on which they base their condemnation of the sedimentation test, are so at variance with the rates obtained in the various gynecologic case types by practically all other workers in this field, as to warrant the impression that their cases were complicated by undetected remote foci of infection or were cases of mixed pelvic pathology. The one article lists carcinoma and uterine fibroids as having identical rates and the other article instances the use of the test seventy-two hours after laparotomy, although it is generally accepted that wound absorption after laparotomy so increases the rate that the test is valueless for one week postoperative. That article also gives comparable rates for fibroids, retroversions and incomplete absorptions.

In the series reported here, the cases were carefully selected, the intention being to prove or disprove the clinical value of the sedimentation test. All patients showing remote active foci of infection, such as tuberculosis, sinusitis, otitis, cholecystitis, cystitis, pyelitis, etc., were eliminated; likewise patients with positive Wassermann reaction or with known history of syphilis. Patients with mixed pelvic path-

ology diagnosed before or during operation, were not considered nor were any whose erythrocytes numbered less than 4,000,000 per cubic millimeter, excepting suspect ectopic pregnancies.

There remained 325 patients answering the above requirements. In each of these, the pathology was proved by subsequent operation, although occasionally the preoperative diagnosis was incorrect. The sedimentation test, however, was always absolutely in accord with the pathology later found on the operating table.

TABLE I

SEDIMENTATION TESTS IN GYNECOLOGY

Cases Under 100 Minutes

| TYPE OF CASE | NO. OF CASES | HIGHEST | LOWEST | AVERAGE |
|---|---|---|---|---|
| Puerperal sepsis | 28 | 45 | 4 | 16 |
| Acute salpingitis | 17 | 42 | 10 | 22 |
| Late carcinoma | 7 | 33 | 9 | 22 |
| Pyelitis | 6 | 45 | 13 | 31 |
| Subacute salpingitis | 16 | 84 | 24 | 56 |
| Early carcinoma | 4 | 115 | 45 | 65 |
| Threatened abortion | 8 | 140 | 40 | 71 |
| Incomplete abortion | 28 | 175 | 40 | 88 |
| Bartholinitis | 4 | 140 | 25 | 82 |
| Miscellaneous | 32 | 95 | 8 | 44 |

TABLE II

SEDIMENTATION TESTS IN GYNECOLOGY

Cases Over 100 Minutes

| TYPE OF CASE | NO. OF CASES | HIGHEST | LOWEST | AVERAGE |
|---|---|---|---|---|
| Ovarian cysts | 7 | 230 | 60 | 113 |
| Chronic salpingitis | 8 | 167 | 52 | 117 |
| Polyps | 5 | 142 | 110 | 123 |
| Endocervicitis | 8 | 255 | 50 | 126 |
| Plastic operations | 41 | 255 | 73 | 135 |
| Fibroids of uterus | 36 | 310 | 75 | 146 |
| Fibrosis uteri | 15 | 240 | 60 | 147 |
| Retroversion | 11 | 300 | 95 | 146 |
| Ectopic pregnancies | 6 | 212 | 45 | 144 |
| Normal women | 12 | 390 | 132 | 184 |
| Miscellaneous | 24 | 274 | 110 | 126 |

The types of cases shown in the tables strikingly demonstrate the value of the sedimentation test as an aid in determining the presence or absence of infection. One hundred and twenty minutes is the generally accepted lower level of normal readings. All the types included in Table I fall below the one hundred minute level, the most acute infections being found at the top of the table, whereas the groups in Table II approximate one hundred minutes or more. In none of these latter cases was an acute infectious process found.

The diagnosis of these group types of cases in Tables I and II must depend primarily on a careful history and physical examination. As a further aid in such a classification, the leucocyte count is of little value and the temperature curve equally valueless except in puerperal

sepsis, acute salpingitis, septic abortions and pyelitis, whereas the sedimentation rate is, by these figures, shown to be distinctly confirmatory of the clinical diagnosis.

Moreover, in each individual patient in whom a differential diagnosis requires considerable consideration, the sedimentation rate can, and frequently in our experience has, aided in establishing the correct preoperative diagnosis. This series includes many patients in whom the sedimentation rate was at distinct variance with the clinical preoperative diagnosis and who at operation were found to have been incorrectly diagnosed, the sedimentation time harmonizing with the actual pathology found. For example:

CASE 1.—Mrs. B., nineteen years of age, para ii. Preoperative diagnosis uncomplicated myoma with bleeding. Sedimentation time 45 minutes (normal average for myomata 146 minutes). The tumor on removal was found to be a chorionepithelioma.

CASE 2.—Mrs. A., thirty-seven years of age, para iv, complained of irregular bleeding, a malodorous discharge and loss of weight. Preoperative diagnosis was malignancy. Leucocyte count 8900, temperature 98, and the sedimentation time 235 minutes. Hysterectomy showed a simple fibrosis uteri.

CASE 3.—Mrs. D., age twenty-six years, nullipara, complained of lower abdominal pain and loss of weight. A mass was palpable in the region of both adnexa. The preoperative diagnosis was probable tuberculous salpingitis. The temperature was 99.2°, the leucocyte count 10,200 and the sedimentation time 165 minutes. Laparotomy revealed normal pelvic organs.

CASE 4.—Mrs. R. N., age thirty-seven years, complained of pain in the left lower quadrant for one year, accompanied by burning and frequency of urination. There was a tender mass palpable in the left pelvis. The temperature was 99°, the leucocyte count was 9300 and the sedimentation time was 134 minutes. The preoperative diagnosis was left tuboovarian abscess. Laparotomy failed to disclose any pelvic pathology.

CASE 5.—Mrs. T., age thirty-one years, complained of abdominal pain, profuse vaginal discharge, with occasional vomiting. Leucocyte count was 13,300 and the temperature 99.8°. There was a tender mass palpable behind and to the left of the cervix. The sedimentation time was 112 minutes. The preoperative diagnosis was pyosalpinx. Operation revealed an adherent retroversion.

The test is of definite value in determining the operability of tubal infections. Every patient with an acute or subacute salpingitis was treated conservatively, with semiweekly sedimentation readings and operated only when the sedimentation time reached sixty minutes or more. With one exception, every one of these patients made an uneventful recovery, the exception running a stormy and febrile convalescense. On the other hand several patients who were operated while the sedimentation time ranged from thirty to forty minutes, had protracted and febrile recoveries, in one instance with a generalized peritonitis.

Too much emphasis cannot be laid on the importance of repeated readings. The clinical picture and the physical findings may seem to

remain unchanged, the temperature curve and leucocyte counts may show no significant variations, while the sedimentation time is changing in direct relation to the changing condition of the patient. This holds good not only in determining safe operability, but even more strikingly in making a prognosis.

At best it is difficult to gauge the outcome of a septic abortion, a puerperal septicemia, or a spreading pelvic peritonitis. We believe that it is safe to make a good prognosis on the strength of a rising sedimentation rate regardless of the severity of the clinical symptoms, hyperpyrexia, or persistent leucopenia and conversely that a falling sedimentation rate is ominous in the face of apparent clinical improvement. For example:

CASE 6.—Mrs. M., age twenty-one years, para i, on the fourth day postpartum developed a temperature of 102.2°, with pulse 136, leucocyte count 10,600 and a sedimentation time of 7 minutes. On the sixth day, the temperature was 102°, pulse 114, leucocyte count still 10,600 and the sedimentation time 12 minutes. On the fourteenth day, the temperature was still 101°, the pulse 100, the leucocyte count 10,000, while the sedimentation time had risen to 18 minutes. On the twentieth day with normal temperature 98.6°, a pulse of 90, the leucocyte count was still 10,200 and the sedimentation rate had risen to 25 minutes.

## SUMMARY

In a series of 325 selected cases each of which showed only one type of gynecologic pathology and no remote foci, the conclusions arrived at in previous publications are confirmed.

1. The sedimentation test is more useful than the temperature curve or the leucocyte count in determining the presence or absence of infection.

2. A sedimentation time of more than two hours rules out infection in the existing pelvic pathology.

3. The test is a further aid in determining the safe time for operation, sixty minutes being the lower limit of safety.

4. The sedimentation test is a more delicate prognostic index, good or bad, than either the leucocyte count or the temperature curve.

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(*For discussion see page 757.*)

# CERTAIN OBSERVATIONS REGARDING PROLONGATION OF PREGNANCY*

By William R. Nicholson, M.D., Philadelphia, Pa.

ONE of the main reasons for presenting a paper upon this subject is that in my early days as an obstetrician I was constantly bothered by the fear of serious results which might follow a prolongation of pregnancy. I, of course, had the experience common to all, that 6-8 per cent of pregnancies went beyond the expected date, but later I realized that for the most part such a fear is without any foundation. For a long time this knowledge has been a comfort to me, knowing as I do, that in the light of our present-day experience it is no longer needful to be alarmed at the mere continuance of pregnancy beyond the calculated date. In making this statement I do not wish to be misunderstood. I do not deny that rarely pregnancy may be prolonged so far beyond the so-called normal limits that if the case be not properly handled there may be very serious results to the child and mother, but what I do affirm is that this true prolongation is really rare and that when it occurs it can be detected in ample time to permit the handling of the case in an intelligent manner and with no more hazard to either child or mother than is a necessary accompaniment to the conduct of labor in the presence of relative degrees of contracted pelvis.

My belief as to the infrequency of prolongation is substantiated by the fact that a careful study of cases reported in the literature as being of this nature, so frequently shows a lack of evidence to substantiate the diagnosis. For instance, in one of the best papers on this subject which can be found in the literature, that of Ballantyne, there is a long list of cases with a synopsis of the salient points in their histories and my first intention was to make a study of these cases for the purpose of this paper, but a very superficial investigation convinced me that it was a useless expenditure of time as most of them were either so imperfectly reported by the original writers that no one could rightly judge of their value, or the facts stated were unsupported by suitable evidence. In other words the acceptance of the unsupported testimony of a patient relative to the date of fruitful coitus, is hardly in accord with the rules of scientific testimony. However, there is no question that occasionally a pregnancy may continue for decidedly longer than the usual period, and it is theoretically interesting to consider the possible causes of such a prolongation. As is well known, not having any knowledge as to what causes labor to start, it is not to be supposed that we will be able to determine the reasons for a prolongation. Various theories, however,

*Read at a meeting of the Philadelphia Obstetrical Society, January 7, 1926.

have been advanced. Most of them can only be considered as foolish; for instance von Winckel thought that the male sex was more likely to result in a prolongation of pregnancy than would be the case if the child were a female. Why any such opinion was ever advanced by such a man, it is hard to realize. Again, it has been thought that absence of the head from the lower uterine segment might be a cause of prolongation. In support of this the known frequency of anencephaly in cases of prolonged pregnancy has been cited. It has also been stated that a woman who has a thirty-day type of menstruation is more likely to have a prolongation than the woman with the usual menstrual interval. One of the later theories for prolongation is founded on the idea that the fetal pituitary gland may play a part in inciting uterine contractions. Those who advance this theory call attention to the above-mentioned relative frequency of anencephalic abnormality, in which case the pituitary gland is generally absent. This belief is of course more in accord with the present-day fashion but is nevertheless purely and simply theoretic.

The *difficulties in diagnosing postmaturity* is another reason for my disbelief in its frequency. From the clinical standpoint the only symptoms which may be considered of value are of course those demonstrable during life, and of primary importance are any which may be found before birth. Those usually given are the weight and size of the child, the condition of the skin and the nerves and the degree of ossification both of the skull and long bones, together with the condition of the placenta, cord membranes and liquor amnii. It is not necessary in this paper to consider the postmortem signs though it may be said that even the postmortem signs are not necessarily proof of postmaturity. In fact, there are only two points which may really be considered before birth; first the size of the child, and second, the degree of ossification in the long bones, as shown by the x-ray. If careful studies have been made throughout pregnancy, and if *pari-passu* with the prolongation beyond the expected date, there is a steady increase in bulk in the size of the baby beyond the usual development, there may be a certain amount of importance attributed to the apparent size of the child. Though it must be remembered that the nutritional process going on in intrauterine life may be markedly accelerated, so that a child at the eighth month is often as large as another baby at full term, or later. With regard to ossification, it is unfortunate that the earlier ideas as to its value as a diagnostic sign of fetal development have been found to be fallacious, but we now know that the x-ray cannot be of as much value in this matter as might have been expected, since it has been determined that the period of appearance of the centers of ossification varies so much that none of them can be depended upon to establish fetal age within the limits of a month.

One of the main questions to be considered is how frequently pro-

longation of pregnancy is met with. The laity in general would, I am sure, affirm that it is a very common condition; one of the bothers of the obstetrician's life being the insistency of nearly all women that they must be going beyond term, when the earliest estimated date of their delivery has just been passed. Many of you have been frequently put to it to convince such patients that they have no cause to be alarmed and, if you believe as I do, to convince them that there is no need of induction of labor, in the great majority of instances. This opinion, namely, that prolongation of pregnancy is an entity frequently met with, is also held by many physicians as an inheritance from remote antiquity and it has been, and in my opinion continues to be, much too prevalent a belief even among many specialists in this branch. I fancy that some of those present can remember many cases seen in consultation, because of the fear on the part of the physician in attendance that the pregnant patient had so far passed term that serious dangers were imminent. As an example of the attitude assumed by some teachers of obstetrics, I may refer to a recent article comprising a series of 150 induced labors in which a prolongation of pregnancy, as evidenced by slight disproportion, was the reason for interference in 65 instances. The writer of this paper remarks that very probably the commonest indication for the induction of labor in the future will be prolongation of pregnancy.

Before an intelligent opinion for or against the frequency of prolongation of pregnancy may be formed, it is necessary of course to determine the normal period of gestation, and secondly what constitutes a true prolongation. We will all agree that we are most densely ignorant as to the number of days which constitute the period of gestation, not only in the human female, but also in the higher mammalia, and I am of the opinion, sharing it with many others, that there is no definite and fixed number of days which can be predicated as the normal. In other words, as I do not believe that a real prolongation is a common happening, neither do I believe that there is, even scientifically considered, a fixed gestation period; certainly there is none for practical everyday use. The reasons for this belief will help in clarifying some of the obscure points of this subject, at least as far as they have a bearing upon practical everyday questions of prognosis and treatment. They may be briefly stated and are as follows:

1. *Inadequacy of reports.* This has been previously considered.

2. *Analogy.* It is found that the cow, whose gestation period is analogous to that of the human female, and in which the date of insemination is controllable, varies in the duration of different pregnancies very decidedly. Thus Spencer found in a series of cases that there was a variation in the period of gestation of 93 days, while Tessier in a series of 446 cows found 19 with pregnancy lasting over 300 days and one in which it lasted 321 days. Of course the date of insemination was

absolutely certain in both instances just cited. Again Krahmer found that the same cow in her successive pregnancies has a very various insemination birth period. Cow 1 varied from 277 days to 286 days in seven pregnancies. Cow 2 from 276 to 283 in seven pregnancies. Cow 3 from 250 to 283 in eight pregnancies. Cow 4 from 280 to 292 in six pregnancies. Cow 5 from 299 to 304 in four pregnancies. Cow 6 from 276 to 295 in four pregnancies. Cow 7 from 275 to 303 in six pregnancies. Cow 8 from 275 to 321 in seven pregnancies.

Now when we consider the duration of pregnancy in the human female by analyzing a considerable series of cases we also find a decided variation. Thus, Reed in 500 cases (last day of flow positive) found that 112 women gave birth between the 281st and 287th day, 63 between the 288th and 294th day, 28 between the 295th and 301st day, 8 between the 302nd and 308th day, 6 between the 309th and 316th day, and 1 at the 301st, 311th, 314th, 315th and 316th day. Von Winckel in a series of 30,500 pregnancies in which the first day of the last period was known and from which the pregnancy was calculated, found 31 cases showing a duration of from 302 to 322 days. Merriman studied a series of 114 pregnancies (last day of last menstrual flow positive) and found 4 in whom pregnancy lasted from 302 to 306 days. Further, if one considers the variable factors with which one has to deal in attempting to determine the gestation period in the human female, one will certainly be convinced that the question of prolongation of pregnancy in any given case is a matter of great uncertainty. We generally can get definite knowledge of the first day of the last period and occasionally the date of insemination (it is to be remembered that spermatozoa may live three weeks in the tubes) but we, of course, can never obtain the date of ovulation or conception. As to conception while it usually undoubtedly occurs within three to four days after insemination and rarely later than the tenth day, there is nevertheless a possible interval of delay which may amount to twenty-three days. All we know about ovulation is that while it usually occurs from the eighth to the fourteenth day of the cycle and is, therefore, most constant just after the flow, it may on the other hand occur at any time during the cycle. It is, therefore, in all probability not unusual for conception to take place just before the first missed period instead of just after the last normal one. In other words, it is to be remembered that when we estimate the probable date of confinement as we calculated from the last period we are not estimating the period of gestation but only the menstruation-birth period, which experience has shown to be the most valuable criterion of the duration of pregnancy available to us, but which actually is but a very uncertain approximation of the actual period of gestation, which, moreover, may itself vary in rather wide limits also. Thus, experience shows that 50 per cent of births occur from the 274th to the 280th day after cessation of the last period, and that most of the re-

maining 50 per cent go into labor between the 260th and 274th day or between the 280th and 294th day, but that in from 4 to 6 per cent of cases pregnancy continues beyond 300 days. If we can obtain the actual date of insemination we find that usually the insemination-birth period may be estimated at 275 days, but naturally such data is in the vast majority of instances unavailable, and even if positive there is still a rather remarkable fraction of error which suggests the question as to whether there can be a postmenstrual conception by a premenstrual spermatozoa. Duncan, in 46 cases with date of conception established, found the average date of labor to be 275 days later. Ahlfeld in 425 cases found it to be 271 days later, Hecker, 273 days later, and Veit in 43 cases found it 276 days later. Now while there is not a very great discrepancy shown in the just-mentioned figures there is certainly a possible suspicion as to the verity of the dates of coition, and this is made more suggestive by the statistics of Nürnberger who, in 206 cases of pregnancy occurring in the wives of soldiers in the late war in whom the date of a single coitus was established without the possibility of error, found that labor occurred from 253 to 297 days afterwards. Another interesting point which may be gleaned from statistics of the recent World War is the relation which a single coitus resulting in pregnancy bears to the period of the menstrual cycle. Thus, Siegel found in a series of 300 one-time cohabitations which resulted in pregnancy, that there were 159 (53 per cent) on the sixth day following the beginning of the last flow; and Nürnberger, in 215 cases, found that in 41.3 per cent cohabition had occurred early in the postmenstrual period, in 9.3 per cent in premenstrual period and in the remaining 49.4 per cent at various times during the interval. In a word, when estimating the probable duration of any pregnancy which has apparently passed well beyond the so-called "term," it is a good practice, in the absence of certain knowledge of the date of insemination, to subtract 23 days from the elapsed time. If, for instance, a woman shows a duration of 300 days, counted from her last period, she may well be only 277 days actually pregnant and, therefore, not a case of prolongation at all.

In the previous résumé of facts enough has been said to emphasize the uncertainty of the calculations upon which in ordinary cases estimation of the age of any pregnancy depends, and as a consequence the foolishness of establishing treatment whether active or passive based upon such necessarily fallacious data must be admitted. A realization of this fact develops the real intent of this paper which resolves itself into a plea for the study of every pregnant woman not alone or chiefly from the standpoint of the subjective history but rather from the standpoint of the physical findings which the individual case may present. That pregnancy may continue until in itself further prolongation is fraught with danger, must be admitted, though, as has been said, such a continuance is in my opinion unusual. Therefore it is essential that one

should be in a position to determine from physical examination whether or not an individual case falls in this category, since the danger of an actual continuation of pregnancy beyond term often results in such an increased size of the child, with special reference to cranial and bi-sacromial diameters and advanced ossification of the skull, that serious results to child and mother are only avoided by skillful treatment. In other words, as was said in the early part of this paper the problem is identical with that presented by a case of relative pelvic contraction. Barbour has said that the head of the child is the best pelvimeter and that is true today and always will be true. In my opinion it is far from necessary that the recent graduate of medicine should be qualified to perform a cesarean section; the student should not even be taught the application of the forceps to the truly high head, but he should be trained, at least during his attendance at prenatal clinics in his interneship, so that he may be able to form an intelligent opinion as to the degree of adaptation between the fetal head and the maternal pelvis. As a result of many years' experience with senior medical students and internes I am forced to the conclusion that those of us who are teaching are falling far short of our responsibility in this regard. While it is true that this estimation of relative size demands experience and practice to enable one to determine the possibilities in the more narrow relationships which may exist between head and pelvis, it nevertheless has been a pleasure to me to observe how rapidly an interne will come to a correct conclusion in the ordinary case after the technic, well-established for this examination, has been demonstrated to him. Nowadays it is a universal practice among experts in obstetrics and also among intelligent general practitioners to study the pregnant woman during the last two months from the standpoint of her pelvic capacity and the relationship existing between it and the fetal presenting part. If such an examination shows a true disproportion between the passenger and the passage the case should be appropriately handled no matter whether she be supposedly a month before term, a month after term, or just at term. The methods of treatment suitable to the varying conditions do not interest me at this time though it may be well to state that it is years since I have felt called upon to induce labor for prolongation of pregnancy either in my private work or in the hospital services under my care; moreover in over 70,000 midwife case reports which have routinely passed through my hands during the last ten years, we have had but six cesarean sections for all causes and no woman has died as a result of forceps or version. We have had one craniotomy and some 60 cases of fetal deaths from forceps trauma, or other injury at birth which could be attributed to oversize of the child. When it is remembered that these operative deliveries are performed in the homes of these patients with no skilled assistance and by doctors of very varying

degrees of experience in this department of surgery, it seems justifiable to me to believe that prolongation of pregnancy in the series just quoted played but a very small part in the dystocia met with. In conclusion I affirm it as my belief that all available evidence supports the hypothesis with which this paper began; namely, that a true prolongation of pregnancy is unusual. On the other hand there is no intention to deny its occasional occurrence. As the induction of labor is not as simple and harmless a procedure as some would have us believe, my plea is for the study of the conditions presented by the individual case taking the history into consideration but subordinating it to the physical findings. It is the ascertained relationship of the presenting part to the fetus which should decide for or against interference rather than any fixed number of days of elapsed time.

2023 Spruce Street. (*For discussion see page* 769.)

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## ABDOMINAL PREGNANCY DEVELOPING AS THE RESULT OF A UTEROPERITONEAL FISTULA FOLLOWING CESAREAN SECTION

By John T. Williams, M.D., F.A.C.S., Boston, Mass.

A CAREFUL search of the literature of abdominal pregnancy and cesarean section has convinced me that the case which I am about to report is unique: namely, a case of abdominal pregnancy developing as the result of uteroperitoneal fistula following this operation.

Mrs. O. C., aged thirty, para iii, was seen November 26, 1922, with Dr. W. H. Nute at the Exeter (N.H.) Hospital. Her first labor four years previously, was operative and resulted in a stillbirth. Second pregnancy was terminated by cesarean section two and a half years previously. Convalescence was normal.

*Present pregnancy.*—Last menstruation occurred January 26, 1922. Confinement was expected November 3, 1922. The patient was seen early in pregnancy by Dr. Nute and advised to have another cesarean at term. She refused to accept this advice and returned to her home in a neighboring town.

Apparently the pregnancy proceeded normally up to October 19, 1922. Fetal movements appeared during the fifth month, and the abdominal development seemed perfectly normal. On October 19, being then within two weeks of the estimated date of confinement, there was a profuse but painless, bloody flow, after which the fetal movements ceased.

A diagnosis was made by the local physician of fetal death in utero. As it was thought that the patient would shortly start up in labor and throw off the dead fetus, a policy of expectancy was maintained. As nothing further happened, however, up to November 20, the patient was sent in to the Exeter Hospital, where she was again seen by Dr. Nute. Believing that the uterus contained a macerated fetus which nature was making no effort to expel, Dr. Nute inserted several bougies through the cervix, apparently into the uterine cavity. No pains whatever followed. The bougies were removed after twenty-four hours. On the fifth day following, the patient's temperature rose suddenly to 102°.

I saw the patient late in the afternoon of November 26. Examination showed

the abdomen distended by a pregnancy at or near full term. No fetal heart could be heard, nor could fetal movements be detected. Fetal parts could be indefinitely outlined, but the presentation and position were not ascertained. The abdominal scar resulting from the previous cesarean was visible just to the left of the median line with its center opposite the umbilicus.

On vaginal examination one finger could be passed through the cervix with difficulty. No presenting part could be reached by the examining finger. On

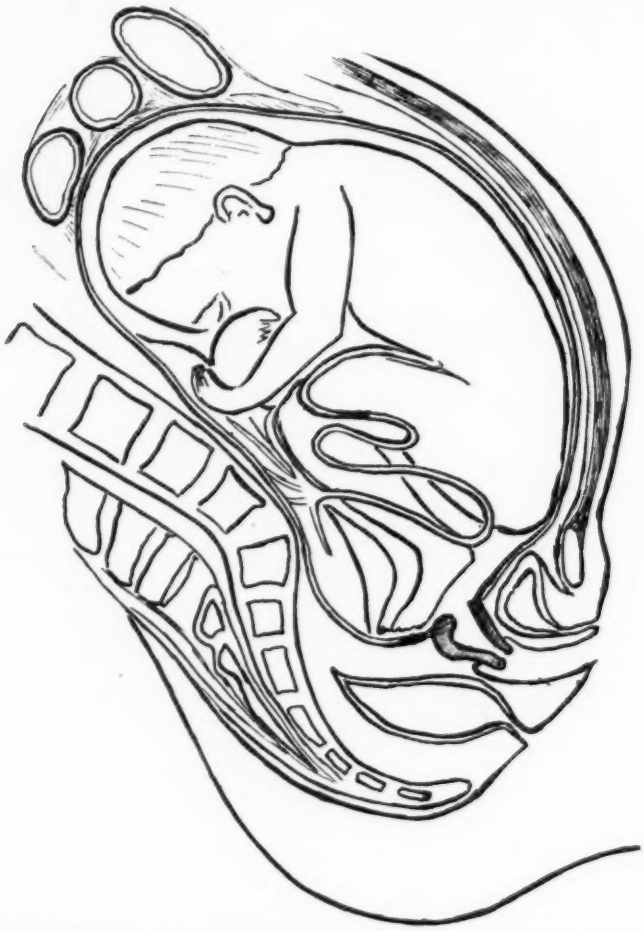

Fig. 1.—Diagram showing abdominal pregnancy. Note uterus (shaded) with fistulous opening on anterior wall near fundus through which the sound, introduced through the cervix, passed into the sac containing the fetus.

withdrawal of the finger there was a gush of dark, slightly foul, fluid blood. A sound was passed through the cervix for a distance of seven inches before meeting with resistance.

It was obvious that manual dilatation was impossible. The attempt to induce labor had already been made and had failed. The choice now lay between vaginal and abdominal cesarean section, and as the presenting part could not even be reached by finger from below, the abdominal route was chosen.

A midline incision below the umbilicus opened into a thin but tough-walled sac containing a macerated fetus of about full term development, presenting by

the breech, S.L.A. The fetus was extracted and the placenta was found densely adherent to the walls of the cavity. It was peeled off with some difficulty.

Exploration of the cavity now showed it to be enclosed by a rather thin but very tough membrane, covering, and adherent to the intestines behind and above, intimately adherent to the abdominal wall in front, and to the peritoneum of the pelvis and iliac fossae laterally. The general peritoneum was not opened. The uterus lay at the bottom of the pelvis, retroverted and imbedded in adhesions, and communicated with the sac by a fistulous opening in its anterior wall, evidently at the site of the former cesarean scar. The appendages were not seen. It was evident that the bougies and sounds, passed from below, had entered the cavity in which the fetus lay, through this opening.

Because of the patient's temperature and the foul condition of the contents of the sac it seemed inadvisable to attempt to dig the uterus out of the tough adhesions in which it was buried. The edges of the uterine fistula were denuded, therefore, and the opening closed with interrupted catgut sutures. The cavity from which the fetus and placenta had been removed was packed with gauze and the abdomen closed, except at the exit of the drain.

This drain was removed gradually, starting on the fourth day, and the patient made a protracted recovery. The temperature remained elevated for nearly two weeks. A profuse foul discharge from the sinus persisted for some weeks, but healing finally took place and the patient eventually returned home in good condition.

I report this case because of its unusual nature. Reports of defective and ruptured uterine scars following cesarean section are not uncommon. In this patient there was no sudden attack of pain and there was no intraperitoneal hemorrhage to suggest a rupture late in pregnancy. The density of the walls of the sac in which the fetus and placenta were enclosed made it obvious that the pregnancy had developed as an abdominal pregnancy at a very early period.

429 Beacon Street.

# Department of Maternal Welfare

CONDUCTED BY FRED L. ADAIR, M.D.

## REPORT OF THE JOINT COMMITTEE ON MATERNAL WELFARE*

DR. ADAIR reported that the representation on the Committee by the American Child Health Association, American Association of Obstetricians, Gynecologists, and Abdominal Surgeons, and the American Gynecological Society, and the personnel, Dr. DeNormandie, Dr. Danforth, Dr. Kosmak, Dr. Lynch, Dr. Lobenstine, Dr. Mosher, Dr. Schwarz, and Dr. Adair, have remained the same as last year. The assignment of states and selection of men in the different states has progressed somewhat. The assignment of states and state leaders is as follows:

Dr. Robert L. DeNormandie: Maine—; Rhode Island—; Massachusetts—; New Hampshire—; and Vermont—.

Dr. W. C. Danforth: Illinois—Dr. Joseph L. Baer; Indiana—; Iowa—Dr. Floyd W. Rice; Ohio; Nebraska—Dr. Earl C. Sage; Wisconsin—Dr. Carl Henry Davis.

Dr. George W. Kosmak: Pennsylvania—; Virginia—; West Virginia—; South Carolina—; Mississippi—; and Connecticut—.

Dr. Frank W. Lynch: California—; Arizona—; Nevada—; Oregon—; Idaho—; and New Mexico—.

Dr. Ralph W. Lobenstine: Delaware—; North Carolina—; New Jersey—; Florida—; New York—; and Maryland—.

Dr. George Clark Mosher: Michigan—Dr. G. Van Amber Brown; Arkansas—Dr. Shelby Boone Hinkle; Missouri—Dr. Otto H. Schwarz; Kansas—Dr. John D. Clark; Oklahoma—Dr. William A. Fowler; and Texas—Dr. Calvin R. Hannah.

Dr. Henry Schwarz: Alabama—; Colorado—; Georgia—; Kentucky—; Louisiana—; and Tennessee—.

Dr. Fred L. Adair: Minnesota—; Montana—Dr. H. A. Tash; North Dakota—; South Dakota—Dr. S. A. Donahoe and Dr. N. T. Owen; Utah—; Washington—Dr. Richard O'Shea; Wyoming—; and the District of Columbia—.

The policy of the Committee has remained the same, namely, that of working with the physicians and surgeons of the country to interest them in raising the level of obstetric practice.

Recently there was published the "Standards of Prenatal Care,"† under the sponsorship of the Children's Bureau. This was worked out by a Medical Committee among the members of which Committee there were quite a number of those from the Committee on Maternal Welfare. The Joint Committee on Maternal Welfare accepts and approves the standards but it is not altogether in favor of the policy of Federal supervision. It is proposed to send out these prenatal standards, not necessarily in government form, but perhaps as a reprint, to all the secretaries of the County, District, and State Societies of the American Medical Association.

The Committee proposes, if it can be done, to proceed with the formulation and publication of some standards of natal and postnatal care and to have them

*Made at the Fifty-first Annual Meeting of the American Gynecological Society, May 21, 1926.

†See issue of June, 1926, page 854.

sponsored by the medical men and societies rather than by the Federal government. To that end Dr. Danforth will prepare the standards on natal care, Dr. Kosmak on postnatal care, and Dr. Lynch will elaborate the prenatal standards which have been published.

This Committee has been financially embarrassed from its inception, but five hundred dollars has been received from the American Child Health Association.

It might be stated that in the very beginning of the work an attempt was made to secure the cooperation of the American Medical Association through the Committeee on Public Health and Legislation but no headway was made. However, in line with Dr. Carl Davis' suggestion of greater and better team work between special societies and A. M. A. sections, it is hoped some cooperation will be forthcoming through the Section of Obstetrics and Gynecology of the A. M. A. As to cooperative activities, it is desired to call attention to the work that is being done in New York State through the members of the profession cooperating with the State Board of Health. The Committee would like to suggest the plan which will be published in the American Journal of Obstetrics and Gynecology for your thoughtful consideration, and if similar plans can be worked out by men from the various states it might be of extreme value. It is felt that we as medical men cannot and should not take the position of hampering health activities on the part of the State and the Federal agencies unless we are prepared to substitute something for them which is equally good or better, and the only way we can accomplish anything constructive is by substitution of our own activities if we wish to curtail the activities of lay and governmental agencies.

For some time some of the nurses have been active in furthering a plan to increase the obstetric training and education of public health nurses, making it possible for them to handle obstetric cases. This is something to which we should give our attention as a Maternal Welfare Committee. Organizations of representative gynecologists and obstetricians should also give these proposals very careful consideration. This problem should be worked out, not alone by nurses and others who are interested, but in conjunction with physicians and not carried too far. These movements should not be national in scope because such activities on the part of nurses are not at all necessary in many communities. It may be the best temporary solution in some localities, but the best ultimate plan is the one for which all should strive. This nurses' and lay organization has asked the help and advice of our Committee and we are of the opinion that it is better to have an entente cordiale with them and try to solve these problems in cooperation with them.

The Rockefeller Foundation, which is interested in a study of maternal hygiene, has also asked our cooperation through one of its representatives, and she is going to make an extensive tour throughout the country looking up the activities of the welfare groups in different communities with particular reference to the effect of their work on maternal mortality and morbidity, and we have been asked by her to furnish the names of medical men in the different states. Your cooperation is solicited in case she calls upon any of you.

There is also the plan for better instruction of nurses in obstetrics which the Society endorsed last year. We propose to formulate an outline for the obstetric education of nurses.

A Committee of the American Pediatric Society, appointed to work out a better classification of the causes of deaths in the newly born, has asked that we cooperate with them in order that there may be a better understanding of these subjects.

A Department of Maternal Welfare is now established in the AMERICAN JOURNAL OF OBSTETRICS AND GYNECOLOGY and it is hoped to make it increasingly attractive and profitable in the future. We are anxious to have criticisms and suggestions as well as suitable contributions from any of you as well as from other interested parties.

## THE TOLEDO, OHIO, ACADEMY OF MEDICINE

### HEARTY HEALTH FOR WOMEN

A lecture course on the above topic has been made possible through the co-operative efforts of The Toledo Academy of Medicine, The Toledo Public Health Association, and The Ohio Department of Health. The program of a popular character consisted of five lectures given in the Academy of Medicine building, from November 8-12. The lectures under the Chairmanship of Dr. R. G. Leland, were as follows:—"Changing Modes and Manners" and "Preparing for Womanhood," R. S. Dixon, M.D., Specialist in Public Health. "Health of Our Mothers," Floyd S. Mowry, M.D., Obstetrician, Western Reserve University, Cleveland, Ohio. "Milestones on the Development and Care of the Child," Wm. M. Champion, M.D., Pediatrician, Western Reserve University, Cleveland, Ohio. "Examination of the School Child"—The Value of Such Health Work to the Child and to the Community, Don W. Gudakunst, M.D., Director, School Health Service, Detroit, Michigan. "More Abundant Health in Middle Life," J. H. J. Upham, M.D., Internist, Columbus, Ohio.

At the close of the lecture discussion by the audience was permitted.

# Society Transactions

## THE AMERICAN GYNECOLOGICAL SOCIETY

### FIFTY-FIRST ANNUAL MEETING

*STOCKBRIDGE, MASS., MAY 20, 21, AND 22, 1926.*

*(Concluded from October)*

DR. JOSEPH L. BAER and DR. RALPH A. REIS, Chicago, Ill., read a paper entitled **Further Studies in Sedimentation.** (For original article see page 740.)

DR. JOHN OSBORN POLAK and DR. VINCENT MAZZOLA, Brooklyn, N. Y., read a paper on **The Clinical Significance of the Sedimentation Test as a Diagnostic and Prognostic Sign.** (For original article see page 700.)

#### DISCUSSION

DR. WM. E. CALDWELL, NEW YORK CITY.—Our experience with the test in gynecologic cases has been limited and we have not as yet reached definite conclusions as to its value. In obstetric cases we always find an increased sedimentation, but believe that this fact is of little importance, either as a diagnostic or prognostic sign. Sedimentation is not greatly changed by the toxemias of pregnancy, although it seems to be decreased when there is serious vomiting. There are so many factors which influence the sedimentation time that the test by itself has not seemed to us of great diagnostic significance.

I was glad to hear Dr. Baer say that the test has been standardized. So many different technics have been described in the literature, and are being used by different clinics, that comparison of the results is impossible.

DR. HARVEY B. MATTHEWS, BROOKLYN, N. Y.—The gravity method in making the test seems to us to be the most accurate. We cannot see how readings can be very accurate when the centrifugal machine is employed. This test is of real value only if taken in connection with other clinical and laboratory diagnostic methods and only if repeated a number of times.

Dr. Polak spoke of the sensitiveness of this test. It is this sensitiveness that makes it of more importance than others we are in the habit of doing. It keeps us from operating on certain cases; it helps us in the differential diagnosis between chronic inflammatory disease and ectopic pregnancy. In the differential diagnosis of certain postoperative complications we have found that the test is of inestimable value. The test is a valuable addition to our modern laboratory methods.

DR. ROBERT T. FRANK, NEW YORK CITY.—No test is perfect and I am the last one to become overenthusiastic about any laboratory test, but I think I am about as nearly enthusiastic regarding this test as any that I have ever come across in medicine. I use the Lintzenmeier technic. Thirty minutes is our rule, and if the sedimentation is below that we do not operate, unless there is some vital indication. In about 150 cases it has proved of utmost value. The weakness of the test is, of course, as pointed out, that other foci, sometimes very difficult to diagnosticate, may interfere with the interpretation of the method.

I would like to call attention to the fact that we have noted in some cases that menstruation appears to favor a short sedimentation time and should be considered as a possible source of error. In one case I have in mind the test proved of utmost value to us. This was a case that had all the earmarks of a pelvic inflammation. The sedimentation time, however, was two hours or over, and I went ahead and found the pelvis filled with Sampson's chocolate cysts.

DR. I. H. NOYES, PROVIDENCE, R. I.—We have recently completed a series of 190 blood sedimentation tests made on 146 different women. In our normal controls the sedimentation time varied from two to four hours or more. In normal pregnancy it diminished as frequency advanced. In two cases of ectopic, one ruptured and the other a tubal abortion with free blood in the pelvis the sedimentation time was 64 minutes and 40 minutes respectively. There were six cases of pelvic abscess, the lowest sedimentation time being 10 minutes, and the highest 25 minutes. Here the inverse ratio between the sedimentation time and leucocyte count remained constant. Inasmuch as a sedimentation time of less than 30 minutes did not always signify a bad prognosis or a protracted recovery, it seemed to us that this test alone should not be relied upon in determining safe time for laparotomy in acute pelvic inflammation, but rather should be considered together with temperature, leucocyte count and clinical findings.

DR. ARTHUR H. CURTIS, CHICAGO, ILL.—Did Dr. Noyes say that he got a short sedimentation time in tubal abortion?

DR. NOYES.—There were two cases of ectopic. One was ruptured, the other, a tubal abortion, with the pelvis filled with blood, which showed a sedimentation time of 40 minutes. The ruptured ectopic with the pelvis also full of blood showed a sedimentation time of 64 minutes. That does not concur with the findings of some other observers.

DR. CURTIS.—That is in contrast with Dr. Baer's observations, is it not?

DR. NOYES.—Yes, it is.

DR. CURTIS.—I would like to ask Dr. Baer to discuss the problem of sedimentation time in tubal pregnancies. There seems to be some discrepancy.

DR. JOSEPH L. BAER, (closing).—I think it well to emphasize at the outset that we are clinicians and not laboratory technicians. We are treating patients and not simply making tests and, of course, the clinical course is the outstanding factor in handling our patients. We use the leucocyte count; we use the thermometer; and now we should use the sedimentation test as well but not place it above clinical observation.

I deliberately introduced into my paper two flat errors in diagnosis to illustrate merely that the sedimentation test in those two cases was a very slow reading, the reading of a normal woman. Nevertheless, the diagnosis had been made in both of these cases and the abdomen was opened and found negative. That was not meant to exalt the sedimentation test above clinical observation, however. In obstetrics we have found it of little value. Of course, the puerperal septicemias fall into the category of rapid readings, but aside from that I believe there is little to be gained from the use of the test routinely in obstetrics.

I must disagree with Dr. Matthews who says that one reading is of no value. One reading that is a fast reading definitely means that there is a focus, and to discard that signal I think is an error.

In tubal pregnancies our tests all show slow sedimentation. These were all ruptured cases, but early before there was a very acute anemia; and we had one reading of 144 minutes. The unruptured case, I think, would show a reading

comparable to that of normal women. There is no infection; there is apparently no agglutinin or globulin disturbance, and no absorption.

As to the value of the test in postoperative cases, in wound absorption there is a definite disturbance in the blood stream and the reading after operation until the acute wound condition has subsided is a rapid reading, quite regardless of what the pathology is or for what the patient was operated upon, and whether she shows a breakdown in the wound afterward. Even with a clean wound after a number of days there is a rapid reading, and I am not sure that that has prognostic and differential value in the early convalescence, so that we have taken the time limit that the European workers have set, which is eight days postoperative, before the test can again be relied upon. Dr. Polak's figures and mine were comparable almost throughout, except that he had for fibroids only 74 minutes, whereas for uncomplicated fibroids we have 36 cases with 146 minutes.

DR. JOHN O. POLAK, (closing).—In regard to that last point, our average normal was lower than Dr. Baer's. In our clinic all of these tests were made by one man. Maybe that explains the constancy of having a lower average for our uncomplicated fibroids than Dr. Baer has. I would say again that the method used in arriving at these conclusions was identical with Dr. Baer's, the modified Lintzenmeier method using the 18 millimeter mark.

Don't let us give you the impression—either Dr. Baer or myself—that we are overenthusiastic, but we do want to leave with you one point, and that is that in addition to our clinical data the proper interpretation of the sedimentation test may save a few women from needless operative procedures. I am convinced of one thing, that in cases of potential septic abortion which we are all treating daily we have in repeated sedimentation tests a positive index, and if it is credited there will be fewer of them interfered with because it warns us of the potential possibility of extending that infection by breaking down nature's barriers.

DR. FRANK W. LYNCH, San Francisco, Calif., read a paper entitled **The Frequency and Meaning of Backache in Gynecology.** (For original article see page 719.)

## DISCUSSION

DR. EDWARD H. RICHARDSON, BALTIMORE, MD.—Some eight years ago I made an intensive study of this subject (*Southern Med. Jour.*, 1918, xl, 139) which was prompted by the failure of operative procedures to cure a definite percentage of cases. This study included not only the clinical side but also a review of both the gynecologic and orthopedic literature of the subject. I reached the conclusion that the actual cause of the backache, in so far as pelvic pathology is a factor, is chronic strain upon the lumbosacral and sacroiliac articulations brought about by faulty posture. Oftentimes this faulty posture results from protective attitudes. These patients naturally assume whatever posture affords the greatest measure of relief from the intrapelvic pain, but in so doing they bring about a defective balance from an orthopedic point of view and, hence, chronic strain upon the joints. I am grateful to Dr. Lynch for showing that congestion must be included as frequent cause of pelvic discomfort, but I believe the associated low back pains are more rationally interpreted as orthopedic in origin. Many of these patients will resume normal postures upon correction of the particular pelvic pathology and their back pains disappear, but there remains a very considerable percentage of cases in which this happy outcome is not achieved until surgical procedures are later supplemented by orthopedic measures to correct faulty posture.

DR. R. R. SMITH, GRAND RAPIDS, MICH.—I believe that most of our difficulty in agreeing on and understanding "backache" and its relations to gynecologic conditions arises from the fact that we have included under this term too great a

variety of disorders and symptoms. We should throw out all diseases of the spine, arthritis, localized or general; strains of the sacroiliac joints; injuries of the spine, sometimes associated with congenital defects of the lower lumbar vertebrae, and the occasional cases of tuberculosis.

The commonest form of backache we see in practice is a *lumbar* backache in the muscles or in muscles and fascia. This form of backache has very little to do with gynecologic conditions. Its most common cause is fatigue. A second cause is faulty posture, which is oftentimes associated with fatigue. Then there are cases of lumbar backache, I believe, due to focal infections. We all have seen some of these cases relieved by the removal of infected teeth or infected tonsils.

A *sacral* backache is a very different thing. It is a referred or reflected pain. A condition in the pelvis causes this pain in the sacrum and it has nothing to do with the muscles or joints. Dr. Lynch emphasizes that he is speaking only of sacral backache. Sacral backache may be caused by gynecologic lesions. I am not able to understand why an uncomplicated ovarian cyst or fibroid, unless they be of large size, should cause sacral backache. A certain proportion of women with normal pelvic organs have pain in the sacrum when they menstruate.

DR. JOHN O. POLAK, BROOKLYN, N. Y.—Dr. Lynch's statement that the majority of sacral backaches were due to pelvic congestion, I believe is very well founded when one realizes the large number of women who have pelvic relaxation and consequently a stasis in the pelvic veins. Furthermore endocervicitis is always attended with some degree of posterior parametritis.

Endocervicitis of varying degree is constantly found postpartum and should be cured before the patient is discharged. When one realizes the immense enlargement of the pelvic vein in every pregnancy, one must appreciate that a routine dealing with this pelvic circulation is going to improve these backaches. We teach our patients from the sixth day to use the knee chest position, and follow that up with the monkey trot for a number of weeks and it is amazing to see the difference in the pelvic congestion of patients so treated and those untreated. We have reduced our postpartal retroversions from 30 to 3 per cent. Lately we have developed the "mule kick," which is something more adaptable to our present housing conditions.

DR. GEORGE GRAY WARD, NEW YORK CITY.—Out of 560 of our cases in which there was backache associated with gynecologic anomalies as far as we could tell, about 80 per cent were relieved by an operative procedure.

The point brought out by Dr. Smith is very important. The interne takes the history and the patient says she has a backache, which may be not the type that Dr. Lynch has referred to at all, or the type that my own study referred to. I believe that there is a definite sacral backache due to pelvic congestion, the result of uterine displacement.

If you relieve in such cases the pelvic congestion by posture, replacement of the uterus, pessary or operation, you will cure the backache.

I found that about 15 per cent of our cases were not relieved by operative procedure, and this led me to establish an orthopedic clinic in the Woman's Hospital. The backache in these cases is found to be due to arthritis, sacroiliac diseases, flat foot, faulty posture or something of that kind. There is a definite need for an orthopedic study in all these cases.

DR. JAMES E. KING, BUFFALO, N. Y.—A short time ago I found that 75 per cent of all women who consulted me came either for relief of backache as their chief symptom or else gave backache as one of their concomitant symptoms. I classify backache in two forms principally. One is the orthopedic form which consists of sacroiliac conditions. The other is the pelvic type and is due to two

causes; one is pelvic congestion as is seen at the menstrual period, the other due to displacements that cause unusual drag on the ligaments. This classifies roughly the types of backache most commonly met. Clinically, women who suffer from any tugging or pulling on the uterine supports will always find relief by lying down. The sacroiliac backache is not relieved by lying down but is often made worse.

It has been rather a surprise in this discussion on backache to hear so little reference made to the Albert Smith pessary. The pessary is one of the most valuable single contributions ever made to gynecology. It not only will relieve the pelvic types of backache but will often aid in differentiation. A backache which is not relieved by a properly adjusted pessary will seldom be helped by surgery. It is very unfortunate that more stress is not laid on the pessary in teaching the medical student. Most students have but vague views and no practical knowledge concerning it.

DR. DOUGAL BISSELL, NEW YORK CITY.—It seemed remarkable that no reference has been made by the essayist to the use of the pessary as a means of relieving backache. I have no doubt that the Albert Smith pessary is of great value but it has always seemed to me that the Emmet pessary conforms to the natural curve of the vagina whereas the Albert Smith's posterior curve is too sharp and strikes the cervix, failing to force it back as far as it should go.

My experience has led me to the conclusion that the faulty position is the result of the physical effort on the part of the patient to relieve the backache and is not the cause of the backache for, when these conditions are found associated and the uterus is replaced and held in proper position with a well adjusted pessary, backache is at once relieved and a correct body position is assumed. There may be exceptional instances, of course, but this is the rule. Many symptoms may result from retroversion but I believe backache is the symptom most commonly met with.

DR. GEORGE W. KOSMAK, NEW YORK CITY.—Many of these women, particularly of the small, thin, asthenic type, who are operated upon for backache in which some gynecologic cause seems evident, have come back with the complaint that the backache is worse. A point not taken into consideration is that to the backache from the gynecologic cause we have added a backache due to a traumatic cause. The woman in a lithotomy position on the operating table will acquire a dislocation of the lower portion of the column, which results in backache often worse than the one due to the original cause, and we should take care to avoid this accident by appropriate cushioning of the table.

DR. FRANK W. LYNCH (closing).—This paper really started as an obstetric follow-up study. We discovered incidentally that we cured 75 per cent of the women who could wear a pessary. The second part of the material for this paper was prepared from a follow-up study of 500 cases, which Dr. Maxwell did, on the function of the ovaries after hysterectomy. As we proceeded with this work we tried to observe the posture of these women. We were familiar with Dr. Dickinson's work, and made silhouettes of these women to see the effect of posture, and we tried to obtain careful histories. We always studied their feet. There are two kinds of flat feet, the congenital type which does not bother them at all, and the acquired. We also take into consideration the flexibility of the spine, ascertained by bending the patient over. Most of these women were young because they came from our obstetric clinic and therefore they were not the type of people who would have ptosis from inflammatory conditions.

We do believe that the series is large enough to convince us, particularly if we take into consideration the experience of the urologists, that the pelvic congestion has a great deal to do with sacral backache.

DR. JOHN A. MCGLINN, Philadelphia, Pa., read a paper on **The Treatment of Granuloma Inguinale with Tartar Emetic.** (For original article see page 665.)

## DISCUSSION

DR. EDWARD A. SCHUMANN, PHILADELPHIA, PA.—This matter of granuloma inguinale brings up so interesting a phase of American medical history that it is well worth a brief review. When I was an interne at the Philadelphia General Hospital, these cases were always to be found in various departments of the hospital and were diagnosed and treated according to the predilection of the particular specialists to whose wards they were assigned. In the department for tuberculosis this disease was termed lupus, in the gynecologic ward it was epithelioma of the vulva and in the venereal service it became syphilis. This went on until 1920, as Dr. McGlinn has stated, when there occurred a case in Bellevue Hospital in which Dr. Douglas Symmers became interested. While studying the case, Dr. Symmers was showing an Argentine medical visitor through the wards, who upon seeing the patient remarked that it was a well developed case of granuloma inguinale. The case was later reported by Symmers and Frost and if I am correct, was the first recorded American incidence of the condition.

The one note with regard to the treatment of granuloma inguinale which I would emphasize is that one should be exceedingly careful to exclude the disease before attempting any operative procedure.

DR. ROBERT T. FRANK, NEW YORK CITY.—The question of granuloma inguinale has interested me greatly since I encountered a case in Colorado. Two operations for carcinoma had been done on this patient in a neighboring state. The condition responded to treatment with antimony but afterward became resistant to it. Because of the severe joint pains resulting from the injections these patients can only stand a certain amount of treatment, and usually return only when they grow worse.

Recently in New York I saw a white patient with what appeared to be a granuloma inguinale. It is surprising that in an adjacent city like Philadelphia cases of granuloma should be so numerous, while in New York there should be so few seen although our attention has been called to it. Other localities have been referred to as endemic centers.

I see no reason to mistake tuberculosis for granuloma. It is not, in my opinion, identical or confusable with granuloma. In the first place, the exuberance of granulomatous tissue is not similar, and it has not the same tendency to spread. Unfortunately, the granuloma inguinale is an uncharacteristic lesion without specific histologic criteria. The Donovan organism has never been proved to be the actual causative factor, because Koch's postulates have never been fulfilled.

I would like to ask Dr. McGlinn in how many of these cases the Wassermann reaction was positive, because luetic infections of the vulva sometimes assume shapes which even practiced dermatologists cannot diagnosticate as characteristic.

DR. FRED L. ADAIR, MINNEAPOLIS, MINN.—I have seen only one case of this sort in all the years I have been on the service at the Minneapolis General Hospital.

The patient gave a history of menopause ten years previously. It was first diagnosed primary carcinoma of the vulva. The diagnosis on biopsy was somewhat obscured by the fact that flies had invaded this growth and there was a good deal of inflammatory reaction. However, an ultimate microscopic diagnosis of granuloma was made by the pathologist and this patient was treated with radium, in 1915. She had a considerable adenopathy in the right inguinal region. The large mass was incised and treated with a radium tube and radium needles were inserted in the groin. Subsequent biopsy showed the recurrent mass to be a squamous-celled carcinoma.

(The doctor showed a second slide with the striking improvement that has taken place. The next slide showed complete healing.) I saw the case just the day before I left Minneapolis, and she showed evidence of a slight recurrence.

DR. WILLIAM P. HEALY, NEW YORK CITY.—The question of the possible similarity between granuloma inguinale and esthiomene has been brought up. There is a very definite clinical distinction. Granuloma inguinale is a painless lesion; esthiomene is an intensely painful lesion. I reported a case of esthiomene, in discussing a paper of Dr. Taussig's two years ago, which was under my supervision for a period of some months. The woman was pregnant and we had to keep her constantly under the influence of morphine.

I have at present two cases of granuloma inguinale under my supervision at the Memorial Hospital. One is a colored and the other a white woman. Radium has helped the colored patient, but only temporarily. We resorted to tartar emetic also with benefit for a time, and then there was recurrence of the lesion. I feel that Dr. Adair has been very fortunate in his case in getting such a complete cure for even such a short period of time. I think that it was possibly largely the result of his surgical treatment of the case.

DR. JOHN A. MC GLINN (closing).—The first case I reported some years ago as tuberculosis of the vulva was shown before some of our postgraduate students. It showed a four-plus Wassermann and was promptly diagnosed by the students as a case of syphilis. Then I tried to impress upon them that because a patient had syphilis was no reason why she should not have another disease in addition to the syphilis. In our cases few have shown positive Wassermann reaction, but the lesion has been absolutely not influenced by antisyphilitic treatment. As Dr. Frank has said, the histologic study of granuloma is not typical because it shows syphilitic granular tissue with a number of leucocytes, the same condition which you find in other tissue.

Referring to Dr. Adair's very favorable result with the use of radium, while on the screen it looks like granuloma, I would almost doubt the correctness of the diagnosis, for in our cases radium has had no influence whatever. In some of our cases, however, x-ray did influence the lesion, but the amount of x-ray necessary to bring about a favorable result is so great that you have to discontinue the x-ray treatment because the skin will not stand a sufficient dosage to bring about any sort of a favorable result.

It has not been proved that the Donovan organism is the cause of granuloma. While you can grow it in broth culture, inoculation experiments have never produced granuloma. Furthermore, implantation of the growth by inoculation of the healthy part of the body has not produced granuloma. We feel, however, that the Donovan organism does possibly play a part because after a second dose of tartar emetic the organisms disappear entirely from the smears and cannot be obtained again.

I feel that so many mistakes in diagnosis have been made in this lesion, and so many operations done or patients condemned to a long period of suffering without something being done, that it is worth while to emphasize the entity of this lesion and its specific treatment.

DR. J. C. LITZENBERG, Minneapolis, Minn., read a paper on **The Relation of Basal Metabolism to Sterility.** (For original article see page 706.)

DISCUSSION

DR. WILLIAM P. HEALY, NEW YORK CITY.—The question of sterility, of course, is one constantly presented to the pelvic surgeon and to the obstetrician for solution, and frequently it is unsatisfactorily solved in the apparently healthy

woman. That is the group to which Dr. Litzenberg, as I take it, refers in his paper, not the definitely endocrine type that is identifiable by outstanding symptoms.

I feel that the ideas presented by Dr. Litzenberg are worthy of very earnest study and consideration. His statistics indicate that undoubtedly by a careful selection of cases some of these women will conceive if appropriate treatment overcomes their low basal metabolism.

DR. HENRY T. HUTCHINS, BOSTON, MASS.—We must remember that a lowered basal metabolism, of course, is not always of endocrine origin; that the metabolic rate may be lowered in a great many ways. It may be thyroid failure, pituitary failure, and also may be due to fatigue or general inanition. Therefore I think Dr. Litzenberg has stated facts correctly when he says that we must regard a low metabolic rate as an *indication of a cause* rather than the cause itself of sterility.

Dr. Lawrence, of Boston, found in his cases of thyroid failure 40 per cent of sterility. In going over his cases he had found two males who had thyroid failure and they both were impotent. But I think in general we must not place all the blame on the endocrines, although there must be something from the endocrine side.

DR. DONALD MACOMBER, BOSTON, MASS.—I have recently thought we are dealing here with a real clinical entity. I believe there are two periods during a woman's life when this entity is particularly called to our attention; one, during adolescence when there is a delay in the appearance of the menses; and secondly, after marriage when there is so apt to be sterility. I believe that this entity is characterized by more than just a low basal metabolism in many instances. In many of these cases we see an increase in the weight over the normal, an amenorrhea of varying length from one to many months, or a scanty menstruation which may be associated with irregular periods. These are not cases of myxedema. They are young women as a rule; they have no mental disturbance; and apart from a slight general inefficiency they have no other symptoms than those mentioned. After the administration of thyroid they almost invariably state that they are more energetic, and are more normal.

In the past year or more, since our attention has been called to this subject, we have had the basal metabolism done on 45 cases where an endocrine disorder was suspected. Of these 20 had a basal metabolism of minus 10. These patients were occasionally of normal weight but usually the weight ran from 20 to 25 pounds and occasionally 35 pounds over so-called normal weight for their age and height. There were no signs of myxedema. Occasionally one would encounter a palpable thyroid. One case was a woman of 18, unmarried, with an infantile uterus. She had a basal metabolism of minus 35. In the married women the uterus was sometimes bordering on the infantile, but usually well within normal limits.

DR. J. C. LITZENBERG (closing).—The "shotgun" glandular tablets of pharmaceutical houses all contain thyroid which is practically the only endocrine which gives any result by mouth, and whatever results there are usually come from the thyroid extract. The use of any of these tablets is inadvisable. The fact that low basal metabolism is not always of an endocrine origin is the reason for my statement that it may be a cause, or the index of a cause. We have plenty of cases of low basal metabolism that may be successfully treated by simple feeding.

This investigation has led us into many bypaths; for instance, we found menstrual disturbances in 40 per cent of these women, which compares well with the 39 per cent of sterilities, indicating that there is a hormonic influence. I hesitated to call this condition hypothyroidism. I don't know what it is, it seems to be an entity, which responds to thyroid or iodine medication. I never give thyroid extract or iodine without a careful study of the basal metabolism. I consider it reprehen-

sible to give thyroid to any case of sterility unless the basal metabolic rate justifies its use, and furthermore the rate must be controlled from time to time so that the medication can be regulated.

DR. ALFRED BAKER SPALDING, San Francisco, Calif., read a paper on **Hemostasis in Vaginal Hysterectomy for Procidentia** (The Ligation of the Lateral Vesicouterine Ligaments for the Control of Hemorrhage in Vaginal Hysterectomy for Conditions of Prolapse of the Uterus with Cystocele). (For original article see page 655.)

DISCUSSION

DR. J. WESLEY BOVÉE, WASHINGTON, D. C.—Hemostasis should be thoroughly practiced but there are different methods of securing it. In doing vaginal hysterectomy for cystocele, prolapsus and procidentia I have followed the plan to secure hemostasis with a modification perhaps of the Mayo procedure, namely of making a T-shaped incision in front of the cervix in the anterior vaginal wall and not separating back far enough on either side to involve the uterovesical ligaments, carrying the incision as near the pubic region as necessary. This is more necessary in the case of the prolapsus of the urethra than in the case where there is no prolapse. Before entering the peritoneal cavity, I first make an opening into the culdesac of Douglas. Dr. Spalding said, I think, that he does that last, but I do it first. Then I open the peritoneal cavity, anteriorly dissecting back the thin flap. Next I bring down the fundus of the uterus put on a broad clamp as close to the uterus as possible including fallopian tube, ovarian ligament, the whole of the broad ligament, the uterosacral and uterovesical ligaments. It may be necessary to use two clamps, but generally one clamp is sufficient on each side. The two sides are brought together by a sewing machine lock stitch so that every part of one side is approximated to the other side. There is none of either lateral stump that is not included in the body of this suture material. Then bringing the upper part of that suture line forward it is sutured at the junction of the urethra with the bladder. If there is a urethrocele the dissection is carried forward so that I can get a bite on the periosteum just a little to the side and behind the pubic arch. Two sutures are placed on either side in this broad ligament shelf that has been constructed securing it anteriorly. Another suture may be necessary. For the two side sutures and the midline one I have used strong chromic catgut. The vaginal mucosa from side to side is united to protect the fascial layers. We have always one weak point here. We have built up a strong shelf anteriorly but we do not know yet, unless we have unusually strong. uterosacral ligaments, whether we shall get a strong pull upward and backward. If these ligaments are torn away form the sacrum, we cannot repair that injury by the vaginal route. This is always a weak point in the vaginal operation.

In the posterior operation the all important thing is to be sure that you get the fascia from the two lateral sides brought in in front of the rectum. Next in importance is to pull the rectum upward to obtain a good pelvic support, provided the uterosacrals are strong. We are going to have some failures in this procedure, although I believe they are small, at least they have been with me, but they are largely due to inefficiency of the uterosacral ligaments.

DR. ROBERT T. FRANK, NEW YORK CITY.—You might be interested in the two cadaver dissections which I made some years ago in which these ligaments can be viewed from above. I would suggest that they be called the pubouterine ligaments because that describes the attachment on both sides. I do not consider myself competent to discuss Dr. Spalding's technic because I do not do vaginal

hysterectomy for prolapsus. I think that if we have preserved the uterus in a prolapse operation in case of recurrence, we are in a much better position to do further work than after the uterus has been removed. I would like to know what the indications are for selecting vaginal hysterectomy. In my material there are a number of cases in younger women where prolapse has occurred and in which I would hate to sacrifice the uterus.

In my cases from 1925 to the present time I found that out of 180 cases of repair, there were 56 cases of prolapse in which 42 ventral fixations were performed and 5 Alexander's. The Alexander operation is done in those cases in which the abdominal wall is unduly flaccid and therefore would allow a certain amount of sagging in the erect posture. In some cases the cystocele predominates, in other cases the rectocele. I am very often able to cure the rectocele rather than the cystocele. In the cystocele I use my own technic. My main object is to preserve the pubocervical (uterine) ligament. I do not separate the bladder as extensively from the fascia as Rawls does. We think it is important to preserve the vascular layer. The fact that all of us have a certain number of recurrences of the rectocele or the cystocele shows that no technic is as yet perfect.

DR. GEORGE GRAY WARD, NEW YORK CITY.—As to the hemostasis: I long ago learned from Dr. J. Riddle Goffe to put a ligature at the base of the uteropubic fascial ligaments to control the bleeding. It is an important point in the technic. Personally I object to the term "high rectocele" because I think in a great many of these cases it is not truly a rectocele but is really an enterocele, a hernia in Douglas' pouch. A failure is very often due to this fact. Therefore my technic in these operations is, after removal of the uterus to dissect out this peritoneal sac. You can then see the uterosacral ligaments and it is not difficult to unite them with linen sutures. By this procedure the liability of a hernia forming from above is eliminated. We have proved this by our follow-up in the majority of cases. We have some failures and those are due, as Dr. Bovée has pointed out, to the fact that the uterosacrals are so weak that they cannot provide sufficient support. Follow-up shows that our percentage of failures is small, and it is always only a small hernia that forms.

The operation of Dr. Spalding procures a proper pelvic floor and particularly takes care of the rectum by lifting it up. I believe I coined the word "rectopexy" for a procedure in which the rectum is lifted up and fastened to the undamaged part of the vagina where the fascia is intact. So far as the anterior vaginal wall is concerned we can cure practically all cystoceles but the the weak point in these operations lies in the fact that the uterosacral ligaments may be attenuated and inefficient as support.

DR. R. M. RAWLS, NEW YORK CITY.—All of us get equally good results in operating for prolapse if our technic is based on an anatomic repair of the injury. In doing operations for prolapse an understanding of the anatomy is essential, especially when dealing with younger women. In the woman well past the menopause extensive dissection is contraindicated and restoration of the anatomic function not necessary. In selected cases of this class I still do a Watkins' interposition operation.

Looking from above into the pelvis we see fascial bands extending out into the base of the broad ligament, and here I would differ from Dr. Spalding and would call these the cardinal ligaments rather than the lateral vesical ligaments. On a lower plane are the secondary lamellae of the pelvic fascia—the vesical or lateral vesical ligaments, the rectal and the rectovaginal layers.

In my operative work I do not make the T-incision for unless one is very careful one is apt to cut the supporting structures. I make an incision from the meatus to the cervix, through the thickness of the anterior wall and demonstrate

the bladder which is pushed off from the underlying tissues by blunt dissection. Next by sharp dissection, for a short distance, on either side the fascia is separated from the vaginal mucous membrane when a definite line of cleavage is reached and the supporting structures are demonstrated. On the posterior wall a somewhat similar method is done in first identifying the rectum. My method in dealing with prolapse is, to do an anatomic repair in women in the childbearing period, a vaginal hysterectomy and anatomic repair in women in the early menopause, and a Watkins' interposition in elderly women or those not physically suitable for extensive dissection.

In high rectocele I appreciate Dr. Ward's work on enterocele—it is now my custom to look for enterocele and where it occurs to obliterate the culdesac and repair the injury to the vaginal vault as well as the injury to the anterior and posterior walls.

DR. JOSEPH L. BAER, CHICAGO, ILL.—Just a word to continue Dr. Rawls' thought. I believe that in most cases of prolapse there is a congenitally deep pouch of Douglas and, therefore, in accordance with what Dr. Ward said, the thing to do is to obliterate the bottom of the pouch.

DR. ALFRED B. SPALDING (closing).—I have never seen the cystocele cured at the time of operation by the Mayo technic, and if it is not cured then I cannot see why you do not always have a recurrence. I do not believe you can cure the cystocele without exposing the bladder, ligating separately the uterovesical ligaments and getting the bladder up out of the way. I do not believe it is possible to prevent recurrence by including in a mass ligature the uterovesical and the broad ligaments and then sewing the stumps under the bladder, because the cystocele is below this point.

In regard to what we do for recurrence after some one has done a vaginal hysterectomy: I do not know of anything more disheartening than to attempt the repair of these cases. I have at times succeeded in making a transverse incision through the scar at the vault of the vaginal wall, separating the bladder again and overlapping the fascia. Unfortunately, the fascia was often found to be atrophied and the result has not been good. One cannot get a good result unless the surgeon has supported the bladder at the time he did the vaginal hysterectomy.

The question has been asked as to when we take the uterus out. We do not take it out if it is very large, because we have found that it is dangerous to do extensive repair work below and then to open the abdomen and do a hysterectomy. I would prefer to do the vaginal work first and the secondary operation some time later.

Dr. Ward's reference to the deep culdesac is an important point. He has shown us how to take care of that properly.

Dr. Rawls spoke about the cardinal ligaments. To my mind there is no such thing as a cardinal ligament. One finds condensations around the blood vessels and around the ureters and nerves but they are not ligaments. I believe in all our plastic work we must use the principle that if we are to improve the posterior support we must suspend the posterior vaginal wall on the broad and the sacro-uterine ligaments.

## OBSTETRICAL SOCIETY OF PHILADELPHIA

*STATED MEETING JANUARY 7, 1926*

DR. BROOKE M. ANSPACH IN THE CHAIR

DR. JOHN C. HIRST, 2ND, read a paper entitled **Suppression of Urine in Connection with Pregnancy.** (For original article see page 673.)

### DISCUSSION

DR. PHILIP F. WILLIAMS.—The inability to void urine in most pregnant women is easily explained by retroversion of the uterus in early pregnancy; in late pregnancy by impaction of the head, and by toxemias from the congestion which occurs during eclampsia. The cases that Dr. Hirst has reported are rather unusual and much more interesting. In the 12 cases of suppression of urine in pregnant women that Rolleston observed, in which autopsy was done, there was a history of previous underlying nephritis, and he does not believe that acute nephritis occurs except in the woman who has had previous kidney disease. In eclampsia where the urine is almost entirely suppressed before death, the kidney picture is much like that of an acute necrosis of the cortex. I recollect two such cases.

DR. GEORGE W. OUTERBRIDGE.—I should like to mention a case showing to what degree kidneys recuperate from an intense toxic condition. Some years ago I had a case of poisoning from a mercurial douche in which the woman was completely anuric for about fifty-four hours and had 260 mg. of blood urea nitrogen. This woman was pregnant and had taken the douche for the purpose of bringing on an abortion, which she accomplished. We decapsulated both kidneys, and after a stormy career with many complications this woman eventually recovered. She was in the hospital from the end of May to the middle of September. (See *Journal American Medical Association*, Jan. 13, 1923.) The interesting feature in this case was that about a year later this woman became pregnant again. I was anxious to see how those kidneys, which had been subjected to that terrific breakdown and anuria for over fifty hours, would now carry on through a subsequent pregnancy. She entered the hospital and had careful blood chemistry studies made, which showed perfectly normal conditions. We sent her in with the idea of interrupting the pregnancy, but this was not done. She had some vomiting, which she eventually got over, and went through the pregnancy satisfactorily and eventually was delivered of an entirely healthy child. She is apparently an absolutely healthy woman today. I think this case is of interest in showing that kidneys that have been subjected to tremendous toxic conditions can recover, in a comparatively short time, to a state where they will carry through in pregnancy.

DR. EDMUND B. PIPER.—The introduction of mercurochrome in the vein, will in the fulminating type of sepsis cause acute suppression of urine. I have reported thirteen such cases followed by death. I don't believe that mercurochrome should ever be given when there is evidence of acute nephritis.

DR. B. C. HIRST.—In these cases one should endeavor to make a diagnosis between apparent and real anuria. That was the problem which presented itself in the cases reported. Apparent anuria is diagnosticated by ureteral catheterization. One may draw a pint of urine out of the pelvis of one kidney in a woman apparently anuric. The commonest cause of a real anuria is mercurial poisoning.

DR. HIRST (closing).—In answer to Dr. Outerbridge's question, the pronounced hydronephrosis on the left side was quite recent. At six months it was not very noticeable, but there was some dilatation in the left pelvis. A month later, after leaving catheter in the left ureter on three occasions, the condition was practically cured when the patient was discharged, the left kidney holding only a moderately increased amount of urine over the normal.

DR. WILLIAM R. NICHOLSON read a paper entitled **Certain Observations Regarding Prolongation of Pregnancy.** (For original article see page 745.)

DISCUSSION

DR. RICHARD C. NORRIS.—This very conservative presentation is to be commended. In France and Austria 300 days is given as the legal limit for full term. In England and this country it is based on a study of the individual case. The trend of modern physiology is that conception really begins during the week preceding the missed period, but the time of expected confinement has been based upon the last menstrual period, a time when we know the woman is not pregnant. Dr. Nicholson should not stress the cow as a standard for studying women. The sex of the newborn cow has been determined very accurately by studies of the maturation of the ovum and regulating the date of impregnation. Not so with the woman whose ovum does not mature in a similar manner and in whom the date of impregnation cannot be regulated. A woman does not, as a rule, go very much over time or fall into labor very much ahead of time, without an explanation for it. There is, however, such a thing as prolongation of pregnancy, and there is such a thing as danger to mother and child from this condition. It was interesting to look over records in the Preston Retreat as related to this subject. In the first 5,000 cases of my service, in which we seldom induced labor for prolongation of pregnancy, there were 140 cases of induction (2.8 per cent). Of these, only 15 were for prolongation of pregnancy; that is once in 333 cases. As my experience grew I found many difficult labors, with high forceps and overgrown babies with extensive lacerations, sometimes partially detached placenta, in the cases permitted to go too long beyond calculated term. I then made it a rule not to permit a case with a floating head to go more than ten days beyond carefully calculated term. In the last 1500 cases we have induced labor 88 times for all causes (5.8 per cent), and 24 times for prolongation of pregnancy, or 1 in 63 cases. Studying the infant mortality in which we induced labor for prolongation of pregnancy, we found a marked improvement. In two cases labor was induced on dead babies, due apparently to prolongation of pregnancy. Just today a woman thought she was at her term, but after careful study of her history we concluded that she was probably about three weeks overdue. Yesterday she was in labor all day and her baby was dead. To deliver the shoulders, amputation of both arms was necessary. The baby's weight was nine pounds and fourteen ounces. That woman had given birth easily at term to other babies weighing 7½ and 8 pounds. One has to make some fixed rule for guidance,—a time beyond which one will not let patients go without the careful special study that Dr. Nicholson has outlined. My rule now is ten days. First, we consider an accurate history as to menstruation and fetal movements. The more uncertain the history the more uncertain is the conclusion in that particular case. Second, as to the size of previous children and the character of previous labors, if a multipara, with the presumption that each child up to the fourth will probably be progressively larger. The size of the pelvis must be accurately known. The stature and size of the father's head. Is the patient an elderly primipara? Is the position occipitoposterior, resting high at the brim, incompletely flexed and with no descent into the pelvis? Is it a breech?

Such cases are prone to prolongation of pregnancy. The oftener I see a woman in the ordeal of labor the more I am convinced that her nervous system is a very important factor as to how her uterus will function and to what extent pain will inhibit uterine function. As some women vary in their ability to digest a meal, so the uterus may vary in its function during labor. The proper functioning of the sympathetic nervous system is of the greatest value. When it fails it may make her obstetrically unfit. Prolongation of pregnancy often occurs in this type. The modern, neurotic, nerve-stressed girl of ultrafashionable life, when labor begins, often goes to pieces, and you have to resort to narcotics and anesthetics early and more often deliver with forceps after helping dilatation by some mechanical means. When this type is allowed to go too much overtime, all these difficulties will be magnified. Now what is the danger of inducing labor for a woman who has gone ten days over her time, in whom you find, after this critical study, some of these conditions to make you believe that she has rather a difficult labor ahead of her? Naturally, infection is thought of as the greatest danger. In all my experience I have seen but two women die from sepsis following induction of labor, and they died after induction of labor done by someone else, and subsequently I was called upon to operate—on one of these a cesarean section. I have never had a patient die after induction of labor done by myself for prolongation of pregnancy. The inherent dangers are dependent entirely upon the one who does it; his technic and the subsequent handling of that case. Once in 300 cases prolapse of the cord has jeopardized the fetus. There is, of course, a greater degree of aseptic technic required in induction of labor and delivery of the patient than in a normal delivery. I think it is often apparent to those who have a wide experience that patients who go ten days or two weeks over the calculated time often have large babies, with large well-ossified heads, with more obstetric complications and more skill required at delivery. I often say, I think if nature permitted the modern woman to have her babies at eight or eight and a half months it would save a lot of trouble. I believe that prolongation of pregnancy has its added dangers. If I have a primipara and she goes ten days overtime, the head not fixed in the inlet, and especially, if she has a high posterior occiput; if she is not obstetrically fit; if her pelvis is on the border line as to size, I usually induce labor by the simplest means at my disposal. I think Dr. Nicholson has overestimated the value of measuring the head with a pelvimeter. I would rather depend upon palpation of the head overlapping the symphysis and the accurate measurement of the pelvis than to hope to reach a conclusion by attempting to push the head into the pelvis.

If the patient goes ten days beyond the calculated period, suppose there has been an error in her menstrual history and suppose we make an error of acting two weeks too soon, the infant will not be endangered. In multiparas there is a wider field for interfering even more promptly. The woman who has had one or two normal labors comes to term with the head riding high above the pelvic inlet, perhaps a posterior occiput, and her pregnancy is prolonged. She gets nervous, her family is nervous, her body chemistry is under stress, she sleeps badly, and daily she becomes less fit, obstetrically, for her labor. It has been as much to my comfort as to my patient to have her enter a well-equipped hospital and, after taking a large dose of castor oil, the following day, under strict aseptic precautions, to dilate her cervix with two fingers, gently detach the lower pole of the fetal sac and give her five minims of pituitrin. The intermittent dilatation is continued for twenty minutes. With few exceptions, labor begins. Anything that makes labor easier for the woman, she deserves to have. Bringing on labor in a hospital properly equipped is not to her detriment, as I have done it over and over again; and as my practice and my experience has grown I have resorted to it more and more. I do not wish to convey the idea that I believe in confinement by appointment: but I believe that, with hospital equipment, if these cases of prolonged pregnancy are so treated, instead of waiting perhaps two or three weeks for Nature, the obstetrician is not taking ad-

vantage, for his patients' real benefit of the good things that have come out of the past and is clinging to what I believe is behind the times.

DR. GEORGE M. BOYD.—I believe that it is exceedingly rare for a woman to go over the normal length of gestation. In cases where the pregnancy seems to be prolonged, it is usually an error on our part brought about by our inability to determine just when conception took place. I agree with Dr. Norris that we should study carefully the patient as she approaches term and note any feto-pelvic disproportion. One objection to the Müller test is that it often necessitates full anesthesia. I do not believe in the routine induction of labor at term as has been recommended. To follow out such a course will invite trouble, for the methods of inducing labor are not always satisfactory. When feto-pelvic disproportion exists, a test of labor is invaluable, for a normal labor with a large infant is apt to be safer than an induced one with a smaller infant.

DR. CHARLES MAZER.—Regardless of whether the unborn baby is postmature or two weeks short of term, if, in our opinion, the head as compared with the inlet of the pelvis is growing too large, it is our duty to prevent damage to mother and baby by inducing labor as near term as possible. Whether we use the classical methods of gauging the size of the head or a method of our own, we can easily foretell a tendency to overgrowth. I induce labor in these cases by giving castor oil, quinine and pituitrin, which is successful in six out of ten cases. In the unsuccessful cases I let nature take her course rather than resort to instrumental induction of labor, because I honestly believe that a version or even a cesarean section is no more hazardous to mother and baby than instrumental induction of labor.

DR. NICHOLSON (closing).—Dr. Piper is perfectly correct when he speaks of the early ossification of the sutures in fontanels and, as I said in my paper, I thoroughly agree that such cases are often not postmature at all. It has been proved that the x-ray will not help in determining the question of maturity, based upon the condition of the sutures in fontanels. I am also thoroughly in accord with Dr. Boyd's disinclination to induce labor without very definite indications. I think it is a grave mistake for any Society such as this to go on record as advocating either the routine induction of labor at term, or the induction of labor without very definite indications, and the whole purpose of my paper was to call attention to the fact that postmaturity of the unborn child is a much more rare condition than is supposed. I fully believe that if the general profession should undertake to induce labor as a routine procedure, there would be a very unfortunate increase of morbidity and mortality. I have no special objection to the surgically trained obstetricians' adoption of the plan of routine induction, though I personally believe it is an uncalled-for procedure, but this is a totally different question when the general profession is advised to interfere in pregnancy, either because the earliest estimated date for the delivery has been passed, or because it is more convenient to the doctor and the patient that labor be brought on at a certain specified date.

DR. CHARLES S. BARNES read a paper entitled **Occipitoposterior Position.** (For original article see page 734.)

DISCUSSION

DR. JOHN C. APPLEGATE.—I am convinced that nothing gives rise to greater traumatism, both maternal and fetal, than the conduct of delivery with the diameters of the head in the pelvis not properly correlated, as in the occipitoposterior position. With regard to the frequency of the occipitoposterior position, I am very sure that 25 to 30 per cent of the cases I see are occipitoposterior positions and

require interference. The next point is the early recognition of this anomalous position. It is not unusual for us to hear that "the forceps have slipped" and nine times out of ten, they have been applied to a head in an unrecognized occipitoposterior position and the tenth is perhaps a hydrocephalic head or some other pathologic condition. I have in mind at this moment a patient whom I saw not long ago where three physicians had attempted to deliver with forceps. The woman had a relatively contracted pelvis and the child was in an unrecognized occipitoposterior position; each physician took a turn and the instruments in each instance failed to hold; the third doctor was a big, husky fellow and he perhaps got a better grip on the head than the others. When he pulled something had to give way, with the result that there was a dead baby, complete laceration of the perineum, and infection of the woman. The early recognition of this condition is most important. The fetal heart sounds, the palpation of the fetal back and small parts are fairly reliable diagnostic signs, but not absolutely reliable; the fontanels and the suture lines are very reliable, although errors arise ofttimes. When labor is not progressing normally I have no hesitancy in introducing enough of the hand to palpate an ear or the nape of the neck, to be sure of the position before any attempt at delivery is made. In regard to the methods Dr. Barnes referred to, manual rotation is a thing that we usually succeed in doing. I have always felt that the lower the head is in the pelvis the easier it is to rotate; if a right occipitoposterior, the left hand or as much of it as necessary, with the thumb down, is introduced, and kept on the right side of the baby's head so that the occiput and thumb rotate forward in the same relative position, pressure at the same time being made on the forehead just above the pubis with the other hand. It is absolutely useless to rotate the head unless you have the assistance of someone to rotate the body of the child at the same time, otherwise it swings back into its original position; then the application of forceps is necessary when spontaneous delivery does not take place. We have applied forceps in the reverse position, but not with the view of rotating the head, but rather to hold the head and rotate it while the body is being rotated. With the application of forceps simply for the purpose of rotating the head, incalculable damage may be inflicted upon the baby. I am always apprehensive about doing version with the occipitoposterior position because it is more difficult to estimate the comparative dimensions between the head and the pelvis in the occipitoposterior position than in the occipitoanterior position.

DR. BARTON COOKE HIRST.—The best method of dealing with occipitoposterior positions, is always to deliver them by the Scanzoni maneuver. It requires only half a turn of the wrist, without the exercise of any force, if the rotation is done at the right time, namely, when the greatest head diameters are out of the bony pelvic outlet and the scalp appears in the partly dilated vulvar orifice. There is a type of contracted pelvis with diminished width in the outlet in which it may be desirable to apply the Scanzoni method in reverse. If one attains skill in this maneuver by practice on the mannikin and the living subject he soon comes to regard an occipitoposterior position as no complication at all.

DR. J. O. ARNOLD.—I am in entire sympathy with the statement, that the more or less frequent occurrence of persistent occipitoposterior positions is no longer a matter of any serious concern because of the ease with which Dr. Hirst can dispose of them. I feel the same way, but I have arrived at my feeling of safety in the handling of these cases, from an experience quite different from that of Dr. Hirst, because I know I can quickly and safely rotate the occiput manually. Manual rotation is a safer method to teach to beginners and to men of limited experience on obstetrics. It is certainly attended with less danger to both mother and child in the hands of those who are not experts, than instrumental rotation, or most other methods that have been proposed.

In a recent review of 400 consecutive deliveries in private and consultation prac-

tice, there were 98 cases of persistent occiput posterior,—almost 25 per cent. This may be slightly higher than the normal percentage of occurrence, because many of these were consultation cases, but I am inclined to think it is not far from correct.

Some years ago, in a paper describing what I styled the "trimanual method of rotating the occiput," I noted that out of 200 deliveries done for other physicians, 50 or one out of every four, were persistent occipitoposterior positions.

This "trimanual method" was successfully used in all those fifty cases, as well as in the 98 of this recent series, and has been routinely used in scores of other cases not included in these groups.

There must, of course, be absolute certainty as to diagnosis and relationships, and then there must be complete anesthesia for a few minutes, which will permit the manual rotation of the head and body at the same time. The hand of the assistant will keep the body in place, and thereby prevent the return of the occiput to its faulty position. I see no reason whatever for the maneuver of Dr. DeLee to keep the head in place by grasping the scalp with a tenaculum forceps.

It is not only unnecessary, but inadvisable to try to grasp the head with the whole hand introduced into the vagina. Simply applying the fingers of one hand to the occipital pole of the head in the vagina, while the fingers of the external hand make suprapubic pressure on the frontal pole—with the assistant swinging the body —will accomplish rotation safely and easily. Any head that will come down into the pelvic cavity, can thus be manually rotated in the pelvis, and one that does not come down, does not belong to the class of persistent occiput posterior, and therefore calls for other treatment.

DR. LEONARD AVERETT.—I very much regret to see that the writer has overlooked the Kielland forceps in the treatment of occiput posterior. You can apply it in any position, which eliminates the manual, or Scanzoni maneuver, and it will not slip.

DR. DANIEL LONGAKER.—This is the class of cases with high fetal mortality, from the ill-judged procedure of forceps application much too early. It requires an unusual degree of skill, and if the parts be not quite dilatable there will surely be a paralyzed or a dead baby. The thing to do is to give some morphia and wait sufficiently long, until the cervix is out of the way and entirely dilated so that it offers no resistance whatsoever, and then do a version. On the basis of between two and three thousand versions done within the last four or five years, I can speak authoritatively on the subject and I think there is no way by which these cases can be so satisfactorily handled. I have used the Kielland instrument with considerable satisfaction.

DR. BARNES (closing).—I believe that the operator should treat the case by that method best adapted to the condition or to the operator's skill. One man can do manual rotation, in most cases, another can more frequently succeed best with forceps rotation.

The point I wanted especially to emphasize was early diagnosis; not to let the case drag wearily on without knowing the condition present. This is often done. It is most important that the diagnosis be made in the first stage of labor, not allowing the case to go unrelieved for an undue length of time, whether relief be given by manual rotation and spontaneous delivery or forceps extraction, or by forceps rotation and delivery, or by version and extraction. The method may depend largely upon the man or probably much upon the individual case.

We all agree that there is room for improvement over present-day obstetrics. I personally do not have quite the equanimity, in dealing with occipitoposterior, that some of my colleagues seem to have. I think that, according to the literature and my observation and yours, there is a large mortality of infants and much maternal morbidity due to this common anomaly.

# Department of Reviews and Abstracts

Conducted by Hugo Ehrenfest, M.D., Associate Editor

## Collective Review

### HYPERCHOLESTEROLEMIA DURING PREGNANCY

By C. F. Fluhmann, M.D., C.M., San Francisco, California

SINCE the development of efficient chemical methods for determining quantitatively the amount of cholesterol present in the blood, great interest has centered on the subject, and during the past fifteen years the medical literature has been flooded with numerous articles on this problem. The many observations that have been reported have brought to light much information on its occurrence in various diseases, on the rôle it plays in infection and immunity, and also on certain pathologic processes which it can itself set up. A great deal remains obscure, however, regarding its exact position in normal physiology, and this especially holds true respecting its presence in unusual amounts during pregnancy. The following review is an attempt to summarize the most important work that has been done on the subject and as far as possible to correlate the findings.

The occurrence of a hypercholesterolemia during the latter months of pregnancy and its disappearance immediately after delivery was first described, in 1911, by Neumann and Hermann,[1] and soon after by Chauffard and his school.[2] This fact has been corroborated by many workers since then, among whom may be mentioned Autenreith and Funk,[3] Klinkert,[4] Huffmann,[5] and Schiller.[6] Other observations also revealed that in this increase both the free cholesterol and the cholesterol-esters take part (Hermann and Neumann,[7] Slemons and Curtis,[8] Bloor and Knudson,[9] Slemons and Stander[10]). Again, it was shown, in 1912, by Hermann and Neumann,[11] and later by Slemons[38] and Slemons and Stander,[10] that this was not an isolated feature, but that it was accompanied by an increase in the blood of closely related substances, namely, neutral fat and lecithin.

#### EXCRETION IN THE BILE

One of the first problems that was attacked by early workers was to determine whether the increase of cholesterol was due simply to a retention in the body or to an increased production. Since normally most of the cholesterol absorbed is excreted in the bile, this was very soon taken into consideration. McNee,[12] in studying three patients who had died during the last trimester of pregnancy, found a very marked increase in the amount of cholesterol excreted in the bile, and hence came to the conclusion that this figure rose with that of the blood values. Chauffard et al,[13] however, demonstrated that ligating the common bile duct in six dogs caused an intense hypercholesterolemia in each case. Later, Bacmeister and Havers,[14] in further experi-

mental work with dogs in which they had produced biliary fistulae, made a series of estimations on the cholesterol content of the bile and found a very considerable decrease in the amount of cholesterol excreted by that channel during the last month of gestation. From this they evolved the theory that the hypercholesterolemia of pregnancy was caused by a retention, a damming back by the liver cells, and not by an endogenous overproduction of the substance. Medak and Pribram,[15] using specimens of bile obtained from human beings by means of a duodenal sound, found that the cholesterol content of the bile diminished from month to month during pregnancy until in the ninth month only very small quantities could be detected. They reported that this fact was also true in those cases of hypercholesterolemia occurring in conjunction with cholelithiasis, nephropathies, hypertrophic liver cirrhosis, icterus catarrhalis and diabetes. This work in regard to pregnancy was further corroborated by E. E. Pribram.[16]

### GLANDS OF INTERNAL SECRETION

It was never considered, however, that the conception of a simple damming back by the liver was a sufficient explanation and there have been a great many attempts to show that cholesterol metabolism is under the control of the endocrine glands. Neumann and Hermann,[1] in their original work, reported the occurrence of a lipoidemia during the menopause, the postclimacteric period, following castration (both in animals and in human beings), and in experimental animals following exposure of the ovaries to x-rays. From this they assumed that in pregnancy there is a functional alteration in the sexual glands which leads to an excessive formation of cholesterol. Although Huffmann[5] felt that menstruation did not influence the cholesterol curve, Shiskin[17] has more recently noted a considerable increase associated with the onset and first two days of menstruation. There is also evidence that the thyroid has some influence on cholesterol, Troisier and Grigaut[18] and later Rémond and Columbiès[19] having demonstrated that thyroidectomy caused a hypercholesterolemia, and Leupold and Seisser[20] were able to lower the blood cholesterol of rabbits by the injection of thyroidin.

But the main interest has centered on the suprarenal and this is particularly important in regard to pregnancy owing to the changes in that gland at this time. Two definite opinions have arisen on this question and they are in direct contradiction. The French school, headed by Chauffard and his coworkers, having noted that the cortex of the suprarenal, of all the tissues of the body, is the richest in cholesterol, have sought to prove that this organ is the most important factor in its production. They claim that cholesterol is actually manufactured by the suprarenal glands and that they are supplemented in this function by the corpus luteum during certain periods, particularly in its regressive phases.[21, 22] In this they have been supported by Albrecht and Weltmann[23] who also believe that increased activity of the suprarenals results in an increase of cholesterol in the blood. Troisier and Grigaut,[18] in support of this theory, showed that unilateral suprarenalectomy in dogs was followed by a transitory hypercholesterolemia after a latent period, and that bilateral suprarenalectomy did not produce this during the short period that the animal survived. Porak and Quinquaud,[24] in studying the cholesterol content of the blood issuing from the suprarenal vein and comparing it with that in the blood of the general circulation (femoral vein,

carotid artery), found considerably more cholesterol in the suprarenal vein, and hence assumed that the difference was due to amounts added to the blood in its course through the gland. The findings in a case of suprarenal adenoma, reported by Yovanovitch,[25] where a high cholesterolemia was present, were taken to indicate an effect similar to a hyperfunction of glandular tissue and thus to strengthen the theory. Laroche,[26] in studies on hypercholesterolemia in nephritis, also supported this idea. In regard to the effect of adrenalin, Wacker and Hueck[27] early reported that in one instance they found a hypercholesterolemia associated with a hyperglycemia four hours following the injection in a rabbit of 4 c.c. of a 1:2000 solution of adrenalin, and Alessandri[28] produced an increase of blood cholesterol in eight out of ten patients half an hour after the injection of adrenalin.

The above views, however, have met with a great deal of opposition, mainly from German experimenters headed by Aschoff and his pupils. Landau,[31] in studying numerous sections of suprarenal glands in various conditions, came to the conclusion that their lipoid content was directly dependent on the lipoid content of the body. That is, the suprarenals do not manufacture cholesterol but act as depositories and hence their content varies more or less according to the amount present in the blood. This was further supported in experiments by Landau and McNee[32, 33] who fed rabbits large amounts of cholesterol and found both a hypertrophy of the suprarenal cortex and an increase in its cholesterol content. Rothschild[34] noted a hypercholesterolemia following unilateral suprarenalectomy, but since he obtained similar results when he removed both glands he opposed the idea that they form cholesterol. Baumann and Holly[35] also came out against the French conception when they were unable to produce any significant change in the blood cholesterol of rabbits by suprarenalectomies. Sternberg[36] has noted a great similarity in the histologic changes of the suprarenal cortex incident to pregnancy and in cholesterol-fed animals, and attributes both conditions to the same primary cause, namely, hypercholesterolemia. Finally, Steinitz,[29] and Joelsson and Shorr[30] report that they have been unable to affect the cholesterol content of blood by injections of adrenalin.

Joelsson and Shorr[30] more recently have studied the subject and also obtained an increase in the blood cholesterol of rabbits following the removal of one or one and a half suprarenals. They are opposed to both the French and German ideas and suggest the possibility that the internal secretion of the suprarenal has some effect on the blood cholesterol similar to that of the pancreas on the blood sugar.

### TOXEMIAS OF PREGNANCY

When in 1913 Bürger and Beumer,[37] and shortly after Autenreith and Funk,[3] each reported a single instance of marked increase of blood cholesterol during eclampsia it seemed as though this might offer a clue to the solution of the problem. Then two years later Huffmann[5] also expressed the opinion that the cholesterol of the blood was particularly increased in eclamptic patients. The question was then taken up by American investigators and Slemons and Curtis[8] found that although the blood cholesterol was increased in a number of cases of eclampsia this was not a distinguishing feature as the increase in some normal pregnant women was still higher. Two cases of pernicious vomiting did not show any increase in the blood cholesterol, and in nephritic toxemias it was found to be normal or dimin-

ished. They then felt that this might be of some use in differentiating eclampsia from nephritis but further studies by Slemons[38] showed this to be incorrect as three cases of eclampsia had normal values. This was corroborated, in 1923, by Slemons and Stander[10] who came to the conclusion that no characteristic change in the blood fat or blood lipoids accompanies the development of eclampsia or allied intoxications of pregnancy. Campbell[39] in a recent very excellent review of the subject of cholesterol suggests that the acidosis found in the latter months of pregnancy is of importance, stating that "it can hardly be coincidence that three such different conditions as pregnancy, diabetes mellitus, and nephritis also tend to produce acidosis and a high cholesterol content of the blood unless there is some connection between these two factors." More recently Baumann and Holly[40, 41] have studied the blood cholesterol in pregnant dogs and rabbits. Here they noted a very different condition from that in human beings, the rabbits showing a 55 per cent decrease which could be accounted for by the cholesterol deposited in various organs, and in dogs there was very little change from the normal. They are inclined to explain the lipoidemia of pregnancy in the human being as a reaction called forth by a mild intoxication, that is, pregnancy in the human being is to be considered on a pathologic basis and in essentially every case causing an intoxication.

### RELATION TO LACTATION

But it was inevitable that the problem should be attacked in the light of conditions peculiar to pregnancy itself, and the relation of cholesterol to lactation must be mentioned first because this is the only branch of the subject in which opposing views and directly contradictory results are not found. It has been shown by Dorlencourt and Palfy[42, 43] that cholesterol as such occurs in small quantities in both colostrum and breast milk and that these amounts vary under different conditions of nursing. In a paper on milk production, Meigs[44] relates how fat is synthesized from the phospholipins of the blood, and owing to the close relationship between lecithin and cholesterol it is quite possible that the latter may also be concerned. Hermann and Neumann[11] considered the mammary gland as a very important factor in the excretion of cholesterol, and found that in women, who did not nurse their babies, the lipoidemia of pregnancy persisted for a longer period after delivery. Slemons and Stander[10] consider that the increase of fat, lecithin, and cholesterol in the blood during the latter part of pregnancy represents a preliminary step in the preparation for lactation. Baumann and Holly,[40, 41] as previously mentioned, found in pregnant rabbits a decrease in the blood cholesterol and phosphatides to nearly half the nonpregnant levels. Moreover, they note that the increase of these substances in the mammary glands and smaller deposits in other tissues, such as the uterus, ovaries, etc., are sufficient to account for the decrease occurring in the blood.

### FETAL METABOLISM

Has the hypercholesterolemia of pregnancy any relation to the fetus? Is it simply a physiologic process to supply the wants of the developing embryo? It has been definitely established that the blood of infants contains much smaller amounts of cholesterol than the mother's blood. This was first demonstrated by Chauffard et al[45] who estimated the cholesterol present in blood from the umbilical

cord immediately after delivery, and it has been corroborated by further studies on the blood of the newborn by many observers, notably Hermann and Neumann,[7] Klinkert,[4] Huffmann,[5] de Simone,[46] Slemons and Stander,[10] etc. Slemons and Curtis,[8] moreover, have pointed out an interesting fact in comparing the blood of mother and infant. They found that free cholesterol existed in the two circulations in approximately the same amounts, the difference being due to the large amount of cholesterolesters present in the maternal circulation and their almost total absence from the fetal blood under normal conditions. From this they argue that free cholesterol may readily pass through the placenta but not cholesterolesters. In addition, as an explanation of the hypercholesterolemia of pregnancy, they suggest that cholesterol may be a waste product from the fetus and so be constantly passing into the maternal circulation and cause the increase noted in the latter months. However, in considering this phase of the problem, Chauffard and his coworkers,[21] although they give no final conclusions, report a series of estimations on blood obtained from the umbilical cord immediately after labor. Here they found that the blood of the umbilical vein (that is, the blood flowing from the placenta to the fetus) is considerably richer in cholesterol than the blood of the umbilical arteries in which the flow is in the reverse direction. Furthermore it is very questionable whether cholesterol *can* pass through the placenta, and if so by what mechanism is it possible? Since cholesterol is a colloid it cannot pass through the placenta according to the laws of diffusion. Gage and Gage,[47] in 1908, injected Sudan III into pregnant rabbits and were unable to demonstrate any of the dye in the fetus, while Mendel and Daniels[48] in experiments with rats found that stained fat when fed to the pregnant mothers could not be recovered in the young. Schönheimer[49] performed a series of experiments in which large amounts of cholesterol were fed to rabbits and guinea pigs, and two of his animals became pregnant. After delivery, the placentae were noted to be very heavily infiltrated with cholesterol, but careful examination of the fetal organs failed to show any excessive accumulation of these crystals, although they were present to the same amount that a normal fetus of that age shows. These facts are taken to indicate that fat cannot pass through the placenta and hence opens up a new problem as to how the growing embryo obtains the fat that it requires. Wesson,[50] in studying this problem, concludes that it is able to synthesize its own fat, and Slemons and Stander[10] suggest that this is possibly from the glucose which is so readily available to the fetus.

## CONCLUSIONS

Owing to the discrepancies in the findings of different workers, it is impossible at present to reach definite conclusions regarding the significance of cholesterol in pregnancy. During pregnancy there is most likely a decreased excretion in the bile; increased activity of certain glands of internal secretion seem to be a factor in its increased production; it does not appear to have any relation to toxic conditions; it probably is not essential for the needs of the fetus, but it seems to be necessary for the production of milk. Further work on the importance of cholesterol in the physiologic economy, its relation to the metabolism of fats and other lipoids (lecithin, etc.) is desirable.

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STANFORD SCHOOL OF MEDICINE.

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# Item

## RESEARCH FELLOWSHIP IN GYNECOLOGY

A fellowship is offered by the Gynecean Hospital Institute of Gynecologic Research of the University of Pennsylvania, to graduates in medicine, who desire to undertake investigations in Gynecologic Research.

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## Erratum

In the article by Dr. Novak entitled "The Significance of Uterine Mucosa in the Fallopian Tube, with a Discussion of the Origin of Aberrant Endometrium" in the October issue, the legends for Figs. 21 and 22 are transposed.

# Books Received

PRINCIPLES AND PRACTICE OF ENDOCRINE MEDICINE. By William Nathaniel Berkeley, One Time Director of the Laboratory of Experimental Medicine, Cornell University Medical College. Illustrated with 56 engravings and 4 colored plates. Philadelphia, Lea & Febiger, 1926.

EMERGENCY SURGERY. The Military Surgery of the World War Adapted to Civil Life. By George de Tarnowsky, Professor of Clinical Surgery, Loyola University Medical School, etc., Illustrated with 324 engravings. Philadelphia, Lea & Febiger, 1926.

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OUR DOCTORS. By Maurice Duplay. Translation and preface by Dr. Joseph Collins. New York, Harper & Brothers, 1926.

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